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Randomized Controlled Trial of Ondansetron vs. Prochlorperazine in Adults in the Emergency Department

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Objective: To compare the effectiveness of ondansetron and prochlorperazine to treat vomiting. Secondary objectives were the effectiveness of ondansetron and prochlorperazine to treat nausea and their tolerability.

Methods: This was a prospective, randomized, active controlled, double-blinded study. Using a convenience sample, patients were randomized to either intravenous ondansetron 4mg (n=32) or prochlorperazine 10mg (n=32). The primary outcome was the percentage of patients with vomiting at 0-30, 31-60, and 61-120 minutes after the administration of ondansetron or prochlorperazine. Secondary outcomes were nausea assessed by a visual analog scale (VAS) at baseline, 0-30, 31-60, and 61-120 minutes after the administration of ondansetron or prochlorperazine and the percentage of patients with adverse effects (sedation, headache, akathisia, dystonia) to either drug. We performed statistical analyses on the VAS scales at each time point and did a subgroup analysis to examine if nausea scores were affected if the patient had vomited at baseline.

Results: The primary identified cause for nausea and vomiting was flu-like illness or gastroenteritis (19%). The number of patients experiencing breakthrough vomiting at 0-30, 31-60, and 61-120 minutes was similar between groups for these time periods; however, more patients receiving ondansetron experienced vomiting overall (7 [22%] vs. 2[3.2%] patients, p=not significant). Nausea scores at baseline and 0-30 minutes were severe and similar between groups; however, at 31-60 and 61-120 minutes, patients receiving prochlorperazine had better control of nausea (24.9 vs. 43.7 mm, p=0.03; 16.8 vs. 34.3 mm, p=0.05). Sedation scores were similar between groups. There were no cases of extrapyramidal symptoms as assessed by the treating physician and there were four cases of akathisia (prochlorperazine=3 [9%], ondansetron=1[3%]).

Conclusion: Prochlorperazine and ondansetron appear to be equally effective at treating vomiting in the emergency department. [West J Emerg Med. 2011;12(1):1-5.]

INTRODUCTION

Nausea and vomiting are common symptoms in the emergency department (ED). Antiemetic agents used to treat nausea and vomiting include phenothiazine derivatives, prokinetic agents and 5-HT_3 receptor antagonists. No agent is used uniformly for nausea and vomiting in the ED and recent studies include comparisons to ondansetron.[1-8] This study compared prochlorperazine to ondansetron for the treatment of nausea and vomiting in the ED.

BACKGROUND

Chemical mediators involved in nausea and vomiting include dopamine, serotonin, histamine, norepinephrine and glutamine.[9-11] These substances activate the chemoreceptor trigger zone (CTZ), found in the area postrema of the fourth ventricle. The CTZ then stimulates the vomiting center, which initiates the act of vomiting. The activation of 5-HT_3 in the gastrointestinal (GI) tract can initiate vomiting by activating the vomiting center. Prochlorperazine and promethazine

Table 1. Baseline characteristics

	Prochlorperazine n=32	Ondansetron n=32	p-value
Age*	41	40	0.50
Female‡	53%	56%	1.00
African-American‡	91%	87%	1.00
Disposition: discharged‡	94%	74%	1.00
Duration of nausea and vomiting (hours)*	186	77	0.09
Vomiting in the emergency department	38%	19%	0.99
Chief complaint: nausea and vomiting‡	69%	50%	1.00

p-values based on *students t-test or ‡chi-squared

are phenothiazine antiemetics that have been used for the treatment of nausea and vomiting secondary to a wide range of pathologies. Their primary mechanism of action is antagonism of D_2 receptors in the CTZ, although phenothiazines also have antihistaminic and anticholinergic properties. The 5-HT_3 antagonist ondansetron blocks serotonin in the CTZ and the gastrointestinal tract. Ondansetron has been examined in over 100 studies for the treatment of nausea and vomiting related to chemotherapy and anesthesia.[10] There are limited data on the use of ondansetron in the ED, except for use in children.[1,2]

METHODS

This was a prospective, randomized, active controlled, double-blinded study designed to compare the effectiveness of ondansetron and prochlorperazine to treat vomiting. Secondary objectives included the effectiveness of ondansetron and prochlorperazine to treat nausea and the tolerability of prochlorperazine and ondansetron in patients with nausea and/or vomiting. Patients were eligible for inclusion if they were admitted to the ED with nausea and/or vomiting. Exclusion criteria included: previous treatment in the ED with antiemetics; missed last menstrual period or pregnancy; less than 18-years-old; conditions with impaired GI tract function (i.e. irritable bowel syndrome); impaired mental status; treatment with antineoplastic agents within seven days prior to randomization; patients unable to read, write, or communicate in the English language; patients leaving the ED against medical advice. Patients were assigned to treatment using a 1:1 random numbers table to 4mg of ondansetron intravenous (IV) push over 2-5 minutes, or 10 mg of prochlorperazine IV administered over two minutes.

Information collected on admission included: demographics, past medical history, social history, chief complaint, suspected cause of nausea and/or vomiting, number of episodes of vomiting prior to randomization, medications prior to admission and during study time period, duration of nausea and/or vomiting (per history), number of episodes of vomiting, and number of dry heave episodes. The presence or absence of extrapyramidal symptoms or akathisia was assessed by physician observation, and no scale or specific criteria were used to rate severity.

Study Outcomes

The primary outcome was the percentage of patients with vomiting at 0-30 minutes, 31-60 minutes and 61-120 minutes after the administration of ondansetron or prochlorperazine. Secondary outcomes included: nausea assessed by a VAS at baseline and three time intervals, 0-30 minutes, 31-60 minutes and 61-120 minutes, based on similar methods by Ernst et al[5]. We measured VAS scores from 0 to 100 mm with zero being no nausea and 100 mm the worst possible score. In addition, we stratified VAS scores into quartile ranges (none, mild, moderate and severe) to aid in the clinical application of the numerical data. Other outcomes included the percentage of patients experiencing adverse effects (sedation, headache, akathisia and dystonia) to ondansetron or prochlorperazine and treatment failures, defined as requiring rescue antiemetic treatment >30 minutes after administration of the study medication. We also assessed sedation and headache by a VAS with scores from 0 to 100 mm (none to most severe) and stratified them into quartile ranges (none, mild, moderate, severe). The need for a rescue treatment was based on physician preference. Rescue medications were not administered ≤30 minutes post administration of study medication. The decision to administer a rescue medication, as well as the medication choice, was up to the discretion of the treating physician and patients were not crossed over to the other treatment group. Blinding could be broken if there was a safety concern; however, this was not required in any patients.

RESULTS

Three hundred fifty-three patients were screened and 64 patients were randomly assigned to either ondansetron or prochlorperazine. Reasons for exclusion consisted of: received prior treatment (38%), refused to participate (19%), history of impaired GI tract function (9%), altered mental status (7%),

Table 2. Proportion of patients in each treatment group with vomiting at 0 to 30, 31 to 60, and 61 to 120 minutes after administration of treatment, in all patients and according to the presence/absence of vomiting at baseline

	Proportion (%) with vomiting at 0-30 minutes	p-value	Proportion (%) with vomiting at 31-60 minutes	p-value	Proportion (%) with vomiting at 61-120 minutes	p-value
All patients						
Prochlorperazine	0/32 (0.0%)	0.49	1/32 (3.2%)	1.00	1/28 (3.6%)	0.36
Ondansetron	2/32 (6/3%)		1/32 (3.1%)		4/32 (12.5%)	
Patients with no vomiting at baseline						
Prochlorperazine	0/20 (0.0%)	1.00	0/19 (0.0%)	1.00	1/19 (5.3%)	1.00
Ondansetron	0/25 (0.0%)		0/25 (0/0%)		2/25 (8.0%)	
Patients with vomiting at baseline						
Prochlorperazine	0/10 (0.0%)	0.13	1/10 (10.0%)	1.00	0/7 (0.0%)	0.19
Ondansetron	2/6 (33.3%)		1/6 (16.7%)		2/6 (33.3%)	

p-values are based on Fisher's Exact Test

GI bleed (5%), unable to obtain IV access (3%), and unable to consent (19%). The primary cause for nausea and vomiting was flu-like illness or gastroenteritis (19%); other causes included hyperglycemia (6%), alcohol intoxication (5%), gastritis (5%), adhesions (3%), cholecystitis (3%), pancreatitis (2%), renal colic (2%), or undetermined (55%). Demographics for the study population are listed in Table 1. The number of patients experiencing breakthrough vomiting at 0-30 minutes, 31-60 minutes and 61-120 minutes is shown in Table 2. Results were similar between groups; however, overall more patients receiving ondansetron experienced breakthrough vomiting [7 (22%) vs. 2 (6.2%) patients, p=0.23]. In addition, one patient randomized to prochlorperazine, and five patients in the ondansetron group required rescue treatment during the study time period (p=0.20). One patient in the prochlorperazine group and four in the ondansetron group received 25mg promethazine IV, and one patient in the ondansetron group received metoclopramide 10mg IV. Results for nausea VAS scores are in Table 3; they were divided into four categories: none (0), mild (1-33), moderate (34-66) and severe (>66). Nausea scores at baseline and 0-30 minutes were severe and similar between groups, however at 31-60 and 61-120 minutes, patients receiving prochlorperazine had significantly lower nausea scores; the prochlorperazine group scores were mild and the ondansetron were moderate at 61-120 min. In a subgroup analysis, there was no difference in nausea scores if the patient had vomited at baseline. Data for the VAS was not complete for all subjects at all time points due to the inability to perform the VAS secondary to increased sedation (prochlorperazine 7 [22%], ondansetron 5 [16%]), or dropouts due to receiving rescue treatment (prochlorperazone 1 [3%], ondansetron 5 [16%]). Sedation scores were mild for both groups throughout the study period and not statistically different between groups, (p>0.05). Headache scores were mild in the prochlorperazine group at all time points, while moderate at baseline and 0-30 min and mild at 31-60 and 61-120 minutes with ondansetron. Headache scores were significantly lower in the prochlorperazine group at all times points, (p<0.05). There were no cases of extrapyramidal symptoms and four cases of akathisia (prochlorperazine=3 [9%], ondansetron=1[3%]).

DISCUSSION

These results demonstrate that prochlorperazine and ondansetron appear to be equally effective at controlling vomiting in patients presenting to the ED with nausea and/or vomiting. Theoretically prochlorperazine may have an advantage over ondansetron, as serotonin can cause the release of dopamine.[12,13] If dopamine is already present, or released, during nausea or vomiting, blocking serotonin will not be able to prevent the action of this dopamine. Although the results were not statistically different, this may explain the improved response with prochlorperazine. Central 5-HT_3 activity may be more associated with nausea considering the benefit of ondansetron in the pre-treatment of nausea associated with chemotherapy and anesthesia, while receptors in the GI tract are important with treatment of emergent vomiting.

When examining the data on antiemetics in the ED, prochlorperazine and promethazine were studied in a randomized double-blind comparison of adults with gastritis

Table 3. Nausea visual analog scale scores for each treatment group, according to time and presence/absence of vomiting at baseline

All Patients	Nausea score at 0 minutes	p-value	Nausea score at 0-30 minutes	p-value	Nausea score at 31-60 minutes	p-value	Nausea score at 61-120 minutes	p-value
Prochlorperazine	78.6 ± 28.5 (n=32)	0.75	47.5 ±33.3 (n=29	0.73	24.9 ±31.8 (n=30)	0.03	16.8 ±29.1 (n=25)	0.05
Ondansetron	72.4 ± 25.6 (n=32)		50.4 ±33.0 (n=30)		43.7 ±33.5 (n=28)		34.3 ±31.7 (n=26)	
Patients with no vomiting at baseline								
Prochlorperazine	70.1 ± 29.9 (n=20)	0.90	44.2 ±32.4 (n=17)	0.73	23.8 ±31.0 (n=18)	0.05	13.4 ±27.5 (n=16)	0.03
Ondansetron	71.1 ± 22.7 (n=25)		47.7 ±30.8 (n=23)		44.4 ±33.7 (n=21)		37.0 ±32.5 (n=21)	
Patients with vomiting at baseline								
Prochlorperazine	88.1 ± 16.4 (n=10)	0.93	50.9 ±38.2 (n=10)	0.38	28.4 ±36.6 (n=10)	0.32	29.0 ±35.0 (n=7)	0.93
Ondansetron	88.8 ± 17.0 (n=6)		68.5 ±32.1 (n=6)		47.3 ±35.1 (n=6)		27.3 ±31.1 (n=4)	

Note: Visual analog scale scores in table are mean ± standard deviation. p-values are based on ANOVA

or gastroenteritis. Patients were well-matched according to gender, age, duration and number of times vomiting, and baseline nausea as determined by a VAS. Eighty-four patients were randomized to IV prochlorperazine 10mg (n=42) or promethazine 25mg (n=42). Patients were then assessed at 30 minutes, 60 minutes and >60 minutes. The results showed that in the prochlorperazine group more patients demonstrated complete relief (determined by patients) within 30 minutes when compared to promethazine (33.4% vs. 16.7%, p=0.021). Results were 50 vs. 47.6% (30 to 60 min) and 16.7 vs. 35.7% (>60 minutes), with prochlorperazine and promethazine respectively. In addition, there were more treatment failures with promethazine compared to prochlorperazine, 13 vs. 4 patients (p=0.03). Two studies examined the effects of ondansetron compared to placebo in primarily pediatric patients admitted to the ED with acute gastroenteritis.[1,2] In the first study (N=107) IV ondansetron resulted in complete cessation of vomiting more often compared to placebo (70% vs. 51%, p=0.04) during ED stay.[1] Oral ondansetron was compared to placebo in 145 patients in the second study.[2] During the ED stay, 87% of patients in the ondansetron group and 64% of patients in the placebo group experienced no emesis (p=0.004). Follow up at 24 and 48 hours showed no difference between groups in the number of episodes of emesis, or proportion of patients with emesis. Recently, ondansetron was compared to promethazine in the ED. One hundred twenty patients were randomly assigned to ondansetron 4mg or promethazine 25mg IV; those receiving prior antiemetics were excluded.[8] Ondansetron and promethazine were found to be comparable, -34 mm vs. -36 mm, respectively, using a 100-mm VAS. In addition promethazine caused more sedation and there were two cases of akathisia in the promethazine group.

LIMITATIONS

Although this study had a strong design there were several limitations. Because we used a convenience sample with a small sample size the results of this study would need to be confirmed in a larger, powered study. We calculated a sample size of 300 patients would be needed to detect a 30% difference in vomiting between groups with a beta of 0.8 and an alpha of 0.05, estimating a 70% effectiveness rate with ondansetron. With a larger study a smaller, yet clinically important, difference in effectiveness may be determined. In addition, a higher percentage of patients in the ondansetron group (26 vs. 7%) were admitted to the hospital. These

patients could potentially be considered at higher risk for nausea and vomiting due to a greater severity of illness and therefore explain the decreased response to ondansetron. All patients were not able to participate in post-treatment assessment VAS scores due to sedation, which reduced the sample size at each time interval. Headache scores were significantly lower in the prochlorperazine group; however, they were also significantly lower at baseline. The differences in scores at baseline could explain the lower headache scores overall in the prochlorperazine group. In this study 4mg of IV ondansetron was used, which is the dose typically used for nausea and vomiting at our institution. However, higher and lower doses of ondansetron have been shown to be effective.[14,15] If a higher dose of ondansetron had been used there could have been improved control of nausea and vomiting with ondansetron compared to prochlorperazine. Finally, our rates of extrapyramidal symptoms or akathisia were low; however, physician self-reporting determined their presence. If a specific scale was used to detect extrapyramidal symptoms or akathisia their rates may have been higher than we reported.[16]

CONCLUSION

Prochlorperazine and ondansetron appear to be equally effective to treat vomiting in the ED. Prochlorperazine may be more effective in controlling nausea. Although this was a prospective, randomized, active controlled, double-blinded study, it had a small sample size and the results should be confirmed in a larger, powered study.

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Punch Injuries: Insights into Intentional Closed Fist Injuries

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Objectives: This study sought to investigate the patterns of injury resulting from a punch mechanism and to investigate the associated psychopathology present in patients with these injuries.

Methods: Retrospective analysis of patients with hand radiographs ordered from the emergency department allowed for identification of patients with a punch mechanism. We recorded injury patterns and queried patients' medical records for associated psychopathology.

Results: 1,292 patients underwent hand radiographs during a one-year time period; 172 patients (13%) were radiographed following an intentional punch injury, identifying 76 fractures in 70 patients. Males contributed a greater proportion of patients presenting with punch injury when compared to females (80% vs. 20%). Males were more likely to sustain fracture from a punch mechanism (48% vs. 11%, OR 7 [95% CI 2.3-20.9]), but were less likely to have preexisting documented psychiatric disease (23% vs. 49%, OR 3.1 [95% CI 1.4-6.7]). Of all fractures, 61% were to the fifth metacarpal, 21% were to the remainder of the metacarpals, and the remaining were fractures to phalanges and bones of the wrist.

Conclusion: Women are less likely to present with punch injury and are less likely to sustain a fracture when they do present but have more associated psychiatric disease. Both men and women presenting with punch injuries have a higher prevalence of psychiatric disease than the background incidence in the population as a whole. Although punch injuries result in a significant number of boxer fractures, a number of other injuries are associated with punch mechanisms. [West J Emerg Med. 2011;12(1):6-10.]

INTRODUCTION

Injuries of the hand and wrist are among the most common traumatic injuries seen in emergency departments (EDs), accounting for up to 15% of all injuries.[1,2] Many of these injuries are fractures, with metacarpal and phalangeal fractures the most common.[2] Although seemingly minor, these injuries have the potential to affect the function of the patient's hand in both the short- and long-term, causing significant morbidity.

Although most hand injuries are unintentional, a large number occur intentionally, as the result of a closed fist striking a hard immovable object. Closed fist or punch injuries are usually associated with fractures of the fifth metacarpal neck (classic boxer's fracture), but this mechanism is also responsible for a variety of other injury patterns. Like other hand injuries, punch injuries have the potential for considerable morbidity. This makes them of even greater significance, as the highest risk population is young males 15-24 years old, and these injuries may impact their ability to work or even permanently disable them.[3,8] Indeed, young men have been found to incur 60% of all hand injuries and more than 90% of all boxer's fractures.[4,9-11]

Substantial psychopathology is believed to be associated with intentional boxer's fractures, including depression, anxiety and personality disorders. In a small study of 14 patients with boxer's fractures, it was found that patients with these injuries have higher levels of anti-social traits, anxiety and self-defeating traits than patients with other types of fractures or normal controls.[12] These patients are also believed to have a high rate of injury recidivism, with studies reporting a repeat injury rate of up to 27%.[4,13]

Although several studies in Europe and the United States have discussed the epidemiology of hand and wrist fractures, most of them are nonspecific regarding the mechanism of injury leading to specific fracture types. In addition, although there have been some studies looking specifically at boxer's fractures occurring from all mechanisms, none have examined the spectrum of bony injuries incurred from a mechanistic point of view, specifically intentional punch injuries. Our objective was to look at injury patterns sustained secondary to a punch mechanism, as we hypothesized that there might be many bony injuries beyond boxer's fractures that occur from intentionally striking a person or object. We also sought to further delineate the presence of psychiatric co-morbidity associated with punch injury and the incidence of injury recidivism in men and women.

METHODS

Study Design and Setting

This is a retrospective study performed at an academic Level I trauma center with an annual volume of 72,000. The study protocol was reviewed and approved by the Institutional Review Board. We included all ED patients who underwent a radiographic study of their hands for intentional punch injuries from July 1, 2007 to June 30, 2008. We excluded children under the age of 13 and patients for whom a confirmatory electronic medical record could not be located. Research associates queried the electronic Picture ArChiving Communication System (PACS) for patients who had hand films performed during the study period and then identified the mechanism of injury through the radiology requisition form and the electronic medical record for that visit. Mechanism of injury was chosen from a closed list of mechanisms (for example, "punch," "fall," "atraumatic," etc). The presence or absence of a fracture or dislocation, the location of the fracture, and the presence of old or healing fractures was noted from the radiology report.

We used the PACS to ascertain if the patient had had previous ED hand radiographs for punch injuries. The electronic medical record was also queried for details regarding the patient's ED visit, including age at time of visit, gender, psychiatric history and final diagnosis. If the radiographic result was unclear, we used the final clinical diagnosis on the chart to determine presence or absence of fracture.

Psychiatric disease was defined as Axis I (major psychiatric disorders), Axis 2 (pervasive personality disorders) and substance abuse disorders listed on the patient's chart either as part of his/her past medical history or listed in the final diagnoses. If a patient was intoxicated, but was not listed as having alcoholism as a medical problem, he/she was not placed under the categorization of possessing a psychiatric disorder. If the patient had an older chart listing mental illness, but none on the current chart being queried, the patient was not placed under the categorization of possessing a psychiatric disorder.

Intentional punch injuries were defined as report by the patient of striking a person or object with a closed fist. If a patient stated he/she was in an altercation but didn't know how the injury occurred, the injury was not counted as an intentional punch injury and was instead categorized as "trauma, other." Additionally, if a patient had a closed fist injury (for example, falling on a closed fist) but was not in the act of striking something or someone, the injury was not categorized as an intentional punch injury, but was categorized by how it occurred (for instance, "fall," or "motor vehicle crash").

We determined injury recidivism by the presence of previous hand radiographs performed for punch injuries in the PACS system. Recidivism was also reported if the patient reported previous punch-related injuries in the setting of old injuries noted on the radiograph from the study period. We did not count reported prior injuries with no radiographic corroboration of injury.

Research associates recorded all data and entered them into a standardized Excel spreadsheet. All items in the spreadsheet were entered from pull-down menus of closed lists. We calculated interobserver reliability among research associates and used descriptive statistics to analyze the data. We used the Fisher Exact Test to determine differences between male and female patients.

RESULTS

During the 12-month study period, 1,292 patients underwent 1,514 hand radiographs from the ED (1.8% of all patients). Four patients had no dictated ED chart and were therefore excluded from further analysis. One hundred seventy-two patients sought medical attention after intentionally striking an object with a closed fist and underwent 192 radiographs. The remaining radiographs were for the following indications: falls (286 films), major trauma (276 films), crush injuries (171 films), other known trauma, such as sports injuries (349 films), rule-out foreign body (86 films), atraumatic hand pain (98 films) and patients with unknown mechanism (52 films). Of the patients with unknown mechanism, seven reported being involved in altercations at the time of the incident, but could not recall how their hands were injured.

Of the 172 patients identified with a closed fist injury, 35 (20%) were women. Of women seen for punch injury, 17

Table 1. Incidence of psychiatric disease and injury in male and female punchers in the emergency department (ED).

	Male n = 137	Female n = 35	Odds Ratio (95% CI)	p-value
Documented psychiatric disease	32 (23%)	17 (49%)	3.1 (1.4-6.7)	< 0.05
Previous ED visit for punch injury	31 (23%)	10 (29%)	-	ns
Previous ED visit for punch injury and psychiatric disease	13 (9%)	9 (26%)	-	-
Previous radiographic fracture	19 (14%)	0 (0%)	-	-
Radiographic fracture	65 (48%)	4 (11%)	7 (2.3-20.9)	< 0.0001
Radiographic fracture and psychiatric disease	15 (11%)	1 (3%)	-	-

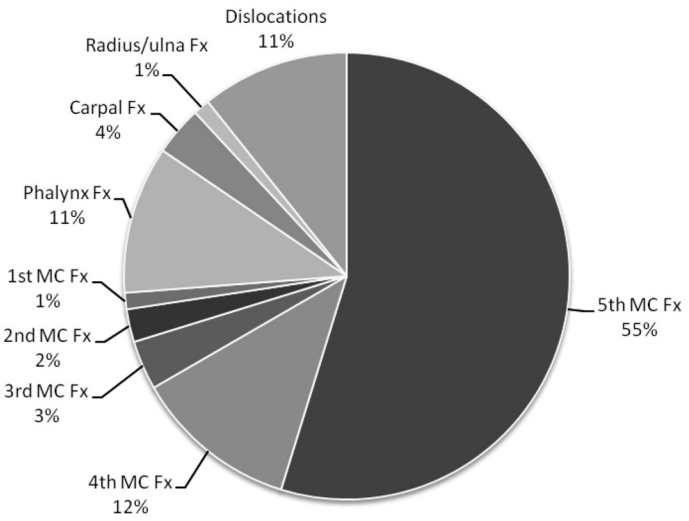

Figure 1. Fracture types occuring from intentional closed-fist injuries.
Fx, fracture; *MC*, metacarpal

(49%) had previous psychiatric diagnoses documented (Table 1). Ten (29%) had been seen in the ED on previous occasions for punch injuries. Four (11%) of the women with punch mechanism sustained a fracture, one of whom had an underlying psychiatric diagnosis. There were no women seen for intentional punch injury who had evidence of old or healing fractures on their radiographs. Ninety percent of female recidivists had documented psychiatric disease on their charts.

Of the 137 male patients evaluated for punch injuries, 32 (23%) had pre-existing psychiatric disease noted on their charts, and 31 (23%) had been radiographed on other occasions for punch injuries. Eleven of these had evidence of old or healing fractures. An additional eight patients had no old studies in the PACS system, but reported previous punch injury and had radiographic evidence of healing or old fractures on their current radiographs, for a total recidivism rate of 28% and a refracture rate of 14%. One-third of these recidivists had a documented psychiatric disease on their charts. Acutely, 65 (48%) of the male patients sustained fractures, and 15 (23%) of these had underlying psychiatric disease.

Males were more likely to suffer fractures from punch injuries when compared to females ($p < 0.0001$) with an odds ratio of 7 (confidence interval 2.3-20.9). Female punchers were much more likely than male punchers to have comorbid psychiatric disease ($p = 0.004$) with an odds ratio of 3.1 (confidence interval 1.4-6.7). There was no difference between the recidivism rate of female and male punchers ($p = 1$) nor was there any relationship between likelihood of fracture and presence of psychiatric disease in either group ($p = 0.69$ for men, $p = 0.60$ for women).

Of the 172 patients imaged for intentional punch injuries, there were 76 fractures in 70 patients (41%). Of all the fractures identified, 46 (61%) were fifth metacarpal fractures; however, the classic boxer's fracture represented only 20 (26%) of these fractures. The remaining were mid- or proximal fifth metacarpal fractures. There were 16 (21%) fractures involving the other four metacarpals. There were 10 (13.3%) fractures of the fourth metacarpal, 3 (4%) of the third metacarpal, 2 (2.7%) of the second metacarpal and 1 (1.3%) of the first metacarpal. In addition, other injuries noted were nine (11.8%) phalanx fractures, three (4%) carpal fractures, and one (1.3%) radius and ulna fracture. There were also nine dislocations noted: three of the fifth metacarpal, three of the fourth metacarpal, one of the third metacarpal, and two involving phalanges (Figure 1).

Interobserver reliability was 1.0 for agreement on diagnosis of punch mechanism, patient age and gender, presence of psychiatric comorbidity, nature of injury, and presence of recidivism.

DISCUSSION

Hand injuries comprise a large proportion of ED visits, representing significant cost to the medical system and potential patient morbidity.[1-2,7-8,11] Most of these injuries are accidental, secondary to falls, crush injuries, or major trauma. However, intentional hand injuries are an important sub-group to consider, since the nature of intentional injuries implies that they are potentially preventable. In our ED, self-inflicted hand injuries accounted for 13% of all hand radiographs performed during the study period. These injuries were almost exclusively to the patients' dominant hand or wrist (one patient fractured both hands), thereby causing additional functional morbidity in terms of the patients' ability to work and perform activities of daily living. The range of bony

injuries was extensive, with only a quarter being the classically described boxer's fracture.

Over the one-year study period, we found that women were far less likely to intentionally punch an object or person compared to men. This finding is consistent with other studies looking exclusively at boxer's fractures from all mechanisms in men and women.[3-4] Women were also less likely to suffer an injury as the result of their punch, with only 11% sustaining a fracture, compared to almost half of men. The smaller number of female punchers and the lack of serious injury in most women after punching an object may be because of differences in male and female baseline characteristics. Past data show that men engage in more risk-taking behavior, suffer more injuries, and harm themselves both fatally and non-fatally more often than women.[14] Our findings may be an extension of these phenomena. This may also be due to last minute hesitation on the part of female punchers, or secondary to the fact that women typically have less upper body strength than men, and do not generate the force necessary to break bone.

Women in this study had a similar overall rate of recidivism (29%) compared to men (23%). These rates are consistent with other studies looking at hand fractures in general and boxer's fractures in particular.[4,13] Interestingly, in spite of this recidivism and the presence of acute fractures in 11% of women, no women had old fractures documented on their radiographs. Admittedly, the sample size of female punchers was small (n=35), but this also suggests that women, in addition to being less likely to suffer bony injury from punching, may also be less likely to do it again when they do incur fractures. Fifteen percent of men imaged for punch injuries had previous fractures documented, which may simply reflect the increased rate of fractures among men or may indicate a difficulty in processing the consequences of this self-destructive behavior.

Nearly half of the women in this study who did intentionally strike a person or object had underlying psychiatric disease, compared to less than a quarter of the men. This is much higher than the incidence of psychiatric disease in the general population, which is estimated to be about 20% for women and 8% for men over the course of a lifetime.[14] Patients with mental illness may be more likely to punch people or objects because of increased frustration or anger related to their illness, poor interpersonal relationships that predisposed to the mental illness, or possibly secondarily to difficulties in verbal communication. Certainly, the disinhibition caused by drug and alcohol abuse may play a role, and many of our patients who had underlying mental illness were also intoxicated. There is a well-described relationship between substance use and abuse and mental illness, and this may play a synergistic role in intentional self-inflicted violent injuries.

Overall, this study demonstrates that punch as a mechanism of injury causes bony hand injuries far in excess of boxer's fractures. Further, it shows that ED presentation for a punch injury is a marker for underlying psychiatric disease. This may seem self-evident to those who practice in emergency medicine, but has not previously been described or documented in the literature. Although our mental health system in the United States is in crisis, these data suggest that it is prudent to screen punching patients for psychiatric stability in much the way we do for those who self-mutilate or present with polysubstance abuse. Although we did not do a financial analysis, there is the potential for considerable cost of these injuries in terms of morbidity and disability to the patient as well as the financial cost to an already overburdened and overcrowded emergency healthcare system. This has been described in other studies.[6-8] Since the rate of recidivism is so high and the co-incidence of punching injuries with psychiatric disease is so common, it seems reasonable to target these patients for urgent psychiatric referral and move toward prevention of these injuries, and not just acute treatment.

CONCLUSION

Self-inflicted hand injuries account for 13% of all hand radiographs performed in the ED. The classic boxer's fracture accounts for only 20% of the fractures sustained through a punch mechanism indicating that a careful search for other injury patterns, including dislocations, is vital in the evaluation of these patients.

One-fifth of the patients presenting for punch injuries are women and although women are less likely to incur fractures from punch injury, about 10% do in fact suffer fractures. Almost half of these patients also carry an underlying psychiatric diagnosis. Men are much more likely to have fractures from self-inflicted injury but have a lower rate of psychiatric illness compared to women. Overall, punch as a mechanism of injury is a marker for underlying psychiatric disease, and it is prudent to screen these patients for this in order to appropriately refer them for care.

LIMITATIONS

This study is limited by nature of its design, which excluded any hand injury that was not radiographed. This could result in potentially missing patients who were transferred to the ED from community hospitals who did not have repeat films on arrival or patients in whom the clinical diagnosis of contusion or fracture was made. Although anecdotally we feel that it is uncommon for a patient to be seen in our ED with a punch-related injury who does not undergo radiographs, it is still possible. Additionally, one could argue that patients who are clinically diagnosed without imaging are likely to possess less physical strength, in order for the clinician to feel there is no fracture, and are therefore more likely to be women. This would result in selection bias. However, we feel the number of missed patients is likely to be small, and although including them would have strengthened our numbers and shown an even bigger impact of these

injuries, it would have made it impossible to determine the specific bony injuries incurred, since none of these patients had films.

In addition, we may have underrepresented the incidence of psychiatric disease in the population, as we only included psychiatric diagnoses mentioned in the past medical history for the visit related to the punch injury, and did not look through previous charts. Therefore, our data is necessarily limited by the thoroughness of the charting of the ED residents, attending physicians, and mid-level providers. These injuries are often cared for in urgent care areas, and therefore may be subjected to less documentation scrutiny. That said, we felt it was more prudent to under-diagnose psychiatric disease rather than erroneously magnify the incidence of co-morbid psychiatric disease. Additionally, we felt that there is a possibility that a patient with psychiatric disease might undergo treatment and no longer be classified as mentally ill, and we did not want to classify "cured" psychiatric patients as having mental illness.

Furthermore, our study relied heavily upon patient history. There were likely many hand injuries from an intentional punch mechanism that were not included because the patient either wasn't sure or wouldn't report what happened. In our dataset, the majority of patients had punched objects, such as trees, walls, or refrigerators. Very few admitted to punching other people. However, some patients had been in altercations, and could not recall how their hands had been injured. All of these patients were excluded from analysis on the basis of unknown mechanism. Again, including these patients would likely have strengthened our argument regarding the importance of these injuries, but we felt the inability to completely classify them from a mechanistic standpoint mandated they be removed from the study population.

Finally, this study was performed at a single academic institution, and the results may not be incorporated to other institutions.

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Genetics of Warfarin Sensitivity in an Emergency Department Population with Thromboembolism

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Background: Emergency department (ED) patients with venous thromboembolism (VTE) are eventually treated with a standard dose of warfarin despite the fact that a number of patients are known to be sensitive to warfarin and may experience supra-therapeutic INRs and adverse bleeding events. Pharmacogenetics is an emerging field of medical practice that seeks to improve drug safety and efficacy in an individual patient by tailoring treatment to the patient's known genetic makeup.

Objective: To identify patients with risk for warfarin sensitivity among an ED population with VTE and to assess if the warfarin sensitivity mutations were of significant enough prevalence to be of clinical significance in customizing treatment of VTE. We sought in a pilot study to identify if testing for common CYP2C9 and VKORC1 single nucleotide polymorphisms (SNPs) in patients who were likely to begin warfarin treatment was feasible in an ED setting.

Methods: A prospective study that identified and enrolled patients presenting to our ED with high clinical suspicion of VTE. Those with high clinical suspicion of VTE were defined as those who had a Doppler ultrasound or computed tomography pulmonary angiography (CTPA) ordered by the primary emergency physician. Blood was taken and processed to ascertain the following SNPs: CYP2C9*2, CYP2C9*3, and VKORC1 3673.

Results: Of the 194 patients enrolled, 132 (68.0%) had at least one known warfarin sensitivity mutation and 114 (58.8%) had the most clinically significant VKORC1 3673 mutation.

Conclusion: A majority of our patients had at least one mutation associated with the atypical metabolism of warfarin. Over half of our population had the most clinically significant VKORC1 3673 mutation. They would likely benefit from individualized warfarin dosing if ever needing anticoagulation. Our initial pilot study shows that allele frequencies of target warfarin sensitivity SNPs in our patient population are frequent enough to make initiation of personalized warfarin dosing feasible. [West J Emerg Med. 2011;12(1):11-16.]

INTRODUCTION

Warfarin sodium is the most popular coumarin anticoagulant used worldwide. In 2004, 31 million dispensed outpatient prescriptions were written for warfarin in the United States (U.S.) alone. This number represents a 45% growth in the number of warfarin prescription from only six years prior and the number continues to rise.[1] Warfarin unfortunately also boasts one of the highest risk profiles for any drug on the market. Its extensive use, along with its high rate of adverse events, adds a significant amount of morbidity and mortality in the U.S. annually. Despite its drawbacks, warfarin remains standard treatment for venous thromboembolism (VTE) because it is effective and inexpensive. The typical emergency physician's use of warfarin is limited to initiating therapy for the occasional low-risk VTE patient who will be treated as an outpatient, as

well as adjusting dosage in the setting of an abnormally high or low INR.

The significant amount of morbidity and mortality attributed to warfarin has made it a favorite drug of focus for the growing pharmacogenetics movement. Pharmacogenetics is an emerging field of medical practice that seeks to improve drug safety and efficacy in an individual patient by tailoring pharmacologic treatment to the patient's known genetic makeup. Pharmacogenetics will undoubtedly produce many novel applications in emergency medicine and personalized warfarin dosing is likely to be among the first. Were emergency clinicians better able to predict a patient's individual response to warfarin treatment when initiating or adjusting therapy, the risk and incidence of adverse effects should be diminished. This knowledge may also serve to increase the proportion of patients with newly diagnosed VTE who are safe to initiate oral anticoagulation at home with close outpatient follow up instead of inpatient warfarin initiation.

The pharmacogenetic focus for warfarin has primarily been on two enzymes involved in its mechanism of action and metabolism: the Vitamin K epoxide reductase complex subunit 1 (VKORC1) and the cytochrome P_{450} 2C9 (CYP2C9) enzyme. We sought to identify in a pilot study if testing for common CYP2C9 and VKORC1 single nucleotide polymorphisms (SNPs) in patients who were likely to begin warfarin treatment was feasible in an emergency department (ED) setting. When researchers utilized VKORC1 mutations, CYP2C9*2, CYP2C9*3, and a number of demographics (age, body weight and concomitant medications) in a Swedish population, they were able to account for 50-60% of warfarin dose variability.[2] By adding knowledge of an individual's Protein C SNPs they were able to increase the dose variation prediction to 62%. Adding on a number of other additional SNPs of minor molecules involved in warfarin's action only increased the prediction to 73%. This showed that while a better prediction of dose variation could be obtained by genotyping many additional SNPs, the majority of warfarin dose variation can be explained by simple patient demographics and the VKORC1, CYP2C9*2, and CYP2C9*3 mutations.[3] As such, the VKORC1 3673 (1639G>A), CYP2C9*2 (430C>T), and CYP2C9*3 (1075A>C) SNPs served as the focus of our investigation. Little is known about the allele frequencies of CYP2C9*2, CYP2C9*3 and VKORC1 3673 in a Hispanic population; we also sought to investigate if the allele frequencies in our predominately Hispanic patient population were high enough to warrant testing for such mutations.

METHODS

This study was an initial pilot study of feasibility. As such we conducted a cross-sectional study with convenience sampling of an ED population with suspected VTE. We chose to study a patient population with suspected thromboembolic disease because we believed VTE diagnosed in the ED would be the most likely time an emergency physician would initiate warfarin therapy. As our selected mutations involve a patient's susceptibility to warfarin sensitivity and not to VTE susceptibility, we did not expect to see any difference in the mutation rates between those patients eventually diagnosed as VTE positive and those VTE negative.

Table 1. Demographics.

		Study Population (n=194)	
Age			
18-29		15	*7.7%*
30-49		77	39.7%
50-69		86	*44.3%*
>70		16	*8.2%*
Average age		50.2	
	Total	**194**	***100.0%***
Gender			
Male		96	*49.5%*
Female		98	*50.5%*
	Total	**194**	***100.0%***
Ethnicity			
Asian		6	*3.1%*
Black		36	*18.6%*
Hispanic		124	*63.9%*
Caucasian		24	*12.4%*
Other		4	*2.1%*
	Total	**194**	***100.0%***

Our study design and protocol was approved by the University of Southern California Health Sciences Institutional Review Board. The authors have no conflicts of interest to report. Trained research assistants identified eligible patients in the LAC+USC Emergency Department and inpatient wards for enrollment. The enrollment time period was from April 2006 to July 2008. Our inclusion criteria were age ≥ 18, ability to give informed consent, in either English or Spanish, and having received an ultrasound of the lower extremities or computed tomography pulmonary angiogram (CTPA) ordered from the ED. Our only exclusion criterion was not satisfying all inclusion criteria.

Trained research assistant obtained informed consent for patients willing to participate, using either English or Spanish documents and consent forms. The study participants were asked a brief focused medical history and to self-identify their ethnicity as White, Hispanic, Black, Asian or other. Blood samples were obtained for each participant. DNA extraction was accomplished using the Qiagen 96 DNA Blood Biorobotic Kit. Genotyping was done using the Taqman 5' Nuclease Assay looking for the following polymorphisms: CYP2C9*2 (430C>T) (rs1799853), CYP2C9*3 (1075A>C) (rs1057910), VKORC1 3673 (1639 G>A) (rs9923231). For each polymorphic site, two oligonucleotide fluorogenic minor

Table 2. Warfarin sensitivity single nucleotide polymorphisms (SNP).

Homozygous wild type (CC)	167
Heterozygous variant (CT)	25
Homozygous variant (TT)	0
Unknown (UU)	2
	194
Variant allele (T) frequency	6.5%
Homozygous wild type (AA)	172
Heterozygous variant (AC)	17
Homozygous variant (CC)	0
Unknown (UU)	5
	194
Variant allele (C) frequency	4.5%
Homozygous wild type (GG)	67
Heterozygous variant (GA)	86
Homozygous variant (AA)	28
Unknown (UU)	13
	194
Variant allele (A) frequency	39.2%

Note: 132 patients of our sample size of 194 had mutations. Some of these 132 had multiple mutations (20 patients with 2 mutations and 2 patients with all 3 mutations). So in total we detected 156 mutations in our 132 patients [(110 patients x 1 mutation) + (20 patients x 2 mutations) + (2 patients x 3 mutations) = 156 mutations].

groove binding probes were designed using Primer Express 2.0 Software (Applied Biosystems). We read and analyzed the fluorescence patterns of the PCR samples using an ABI 7900HT Sequence Detection System and Sequence Detection Software (Applied Biosystems). For reporting purposes, the CYP2C9*2 and *3 haplotypes are often combined in the literature and written as *1/*1 (for wild type), *1/*2, *1/*3, *2/*2, *2/*3, and *3/*3.

RESULTS

Of the 194 patients enrolled, 132 (68.0%) had at least one warfarin sensitivity mutation of either the CYP2C9 enzyme or VKORC1 receptor (Table 2). We had 20 patients (10.3%) with two mutations and two patients (1.0%) with all three mutations. For the most clinically significant VKORC1 3673 (1639G>A), we had 114 patients (58.8%) with mutations, 86 (44.3%) of whom were heterozygote variants (GA) and 28 (14.4%) of whom were homozygote variants (AA).

Table 3. Allele frequencies by ethnicity.

	CYP2C9*2 (T)	CYP2C9*3 (C)	VKORC1 3673 (A)
Hispanic Cohort (n=124)	***5.7%***	***3.3%***	***46.6%***
Reported in the Literature:			
Asian[4-6]	0%	1.5 - 2.8%	89.9 - 91.4%
Black[7,8]	5.2%	1%	8.8 - 9.5%
Hispanic[9-12]	4.8 - 12%	3 - 6.0%	45%
White[3,4,7,8,13-15]	10.5 - 13.1%	5.8 – 8.4%	37.1 - 43%

Our study also offered a unique opportunity to test the allele frequencies of a predominantly Hispanic cohort of patients. Approximately 64% (n=124) of our population self-identified as Hispanic. Calculated variant allele frequencies for our Hispanic cohort were: 5.73% for CYP2C9*2 T allele, 3.28% for CYP2C9*3 C allele, and 46.64% for VKORC1 3673 A allele (Table 3).

DISCUSSION

In 2002 and again in 2004-2005, warfarin and insulin were the two most commonly identified medications associated with adverse events presenting to U.S. EDs.[16-18] From 1999-2003 there were an estimated 29,000 ED visits in the U.S. for warfarin bleeding complications alone. Throughout the 1990s and 2000s warfarin has continually been in the FDA's Adverse Event Reporting System's top 10 most reported drugs.[1] The frequency of "major bleeding" (leading to death or requiring hospitalization or transfusion) adverse effects for patients on warfarin varies by study but has been reported to be anywhere from 0-16%.[19-21] From a survey of U.S. death certificates, anticoagulants were the most frequently mentioned drugs causing "adverse events in therapeutic use" in 2003 and 2004.

The pharmacogenetic focus for warfarin on CYP2C9 and VKORC1 has been because these two enzymes are key to warfarin's mechanism of action and metabolism. Normally, Vitamin K is recycled by the liver using the VKOR enzyme complex. This allows for the efficient use of Vitamin K and allows for sufficient stores of the reduced forms of the Vitamin. Reduced Vitamin K is the main cofactor used by gamma-glutamyl carboxylase (GGCX) to activate Vitamin K-dependent proteins: factors II, VII, IX, and X and proteins C and S. Warfarin acts by blocking the VKORC subunit 1 (VKORC1), thereby preventing the recycling of Vitamin K. This leads to a depletion of the body's Vitamin K stores and decreased activation of Vitamin K-dependent clotting factors. Inactivated Vitamin-K dependent proteins are eventually secreted from the body.[3] Warfarin sodium, as administered, generally consists of a racemic mixture of the R-enantiomer and S-enantiomer of the drug. The S-form is 3-5 times more active than the R-form.[22]

Once in the liver, S-warfarin is primarily metabolized by the cytochrome P_{450} 2C9 (CYP2C9) enzyme.[3] While other cytochrome P_{450} enzymes metabolize R-warfarin, since it is the less active form, these enzymes are not as clinically significant to the pharmacokinetics of warfarin.

Numerous researchers have explained up to 50-60% of the variance in warfarin doses seen among patients, using the common VKORC1 and CYP2C9 SNPs and known environmental factors.[2,14,15,23-28] Mutations in the VKORC1 gene alone are reported to account for approximately 25-30% of warfarin dose variance.[2,15,29] CYP2C9*3(1075A>C) mutation is a SNP substitution of a normal adenine with a cytosine at nucleotide position 1075 of the gene. This mutation severely impairs the CYP2C9 enzyme from hydroxylating s-warfarin, leading to a 27-fold decrease in enzyme efficiency and 71-97% reduced warfarin clearance.[4,30] The CYP2C9*3 mutation alone contributes approximately 10-12% to warfarin dose variance.[3,15,29,30] The CYP2C9*2(430C>T) mutation is a SNP substitution of a normal cytosine for thymine at nucleotide position 430 of the gene. This SNP also results in reduced warfarin metabolism, but only an approximate six-fold reduction in enzyme efficiency and 29-58% reduced warfarin clearance.[4,31,32]

The specific CYP2C9*2 SNP contributes only 2.5% to warfarin dose variance.[2] However, many other CYP2C9 SNPs are in strong linkage disequilibrium with CYP2C9*2, and testing for the *2 SNP likely continues in the literature because it represents the best way to test for all the other SNPs in linkage disequilibrium with it. When researchers tested 55 other CYP2C SNPs, only one added more to dose variation prediction than simply using CYP2C9*2 and CYP2C9*3 alone.[3] So, testing of the CYP2C9*2 and *3 SNPs remains a good way to approximate an individual's CYP2C9 phenotype.

There are an increasingly large number of newly discovered mutations of the VKORC1 receptor. One of the most commonly studied mutations is the VKORC1 3673(1639G>A). This SNP substitutes a normal guanine with an adenine at nucleotide position 1639 of the gene. Because this SNP has been found to be in strong linkage disequilibrium (r^2>0.9) to a number of other VKORC1 SNPs significantly associated with warfarin dosing (381, 6484, 6853, and 7566) it is a good candidate for testing all of these mutations.[29] It is believed that individuals with mutation VKORC1 enzymes have lower baseline enzyme activity and therefore lower baseline levels of active Vitamin K-dependent clotting factors.[33] These individuals would already be predisposed to spontaneous bleeding, and the addition of an "average" warfarin dose could push these individuals to supra-therapeutic INR levels. In a study by Sconce et al[14], those with the homozygous variant mutations for VKORC1 3673 required only 2.23 mg/day warfarin versus the heterozygote variants averaging 3.83 mg/day and homozygous wild type averaging 4.53 mg/day.

It is interesting to note that at least 68% of our patient population at risk for warfarin adverse effects had at least one warfarin sensitivity mutation of either CYP2C9 or VKORC1. Further, 58.8% had the VKORC1 mutation, which is known to be the most clinically significant contribution to warfarin sensitivity. This means that at least this 58% of our population would likely have benefited from a decreased initial warfarin dose than standard algorithms would have prescribed. Although our study specifically looked at VTE patients, we would expect to find similar mutations rates for all patients needing warfarin anticoagulation (atrial fibrillation, prosthetic heart values, pre-op patients, etc.). However, mutation rates for these specific populations need further testing before such assumptions can be verified. Our pilot study also showed that testing for warfarin sensitivity SNPs in the ED for patients prior to starting the medication may be feasible once more rapid testing is widely available. The allele frequencies of the VKORC1, CYP2C9*2, and CYP2C9*3 in our population were high enough to warrant testing for these mutations.

Our study also helped add to the knowledge of VKORC1 3673, CYP2C9*2, and CYP2C9*3 allele frequencies in a Hispanic-American population. Review of the literature revealed only three prior studies looking at Hispanic CYP2C9 allele frequencies and one additional study to include VKORC1 allele frequencies in a Hispanic population.[9-12] Two studies looked at South American populations and found allele frequencies of 4.8% for CYP2C9*2 and 3.0% for CYP2C9*3 in 778 Bolivians and VKORC1 3673 frequencies of 45% for 191 genetically isolated Columbians.[9,11] Another study looked at 434 Hispanic-Americans and found allele frequencies of 12.0% for CYP2C9*2 and 3.4% for CYP2C9*3.[12] Finally, one study specifically looked at the allele frequencies differences between Mexican-Americans and Spaniards and found that 196 Mexican-Americans had allele frequencies of 8.0% for CYP2C9*2 and 6.0% for CYP2C9*3; these frequencies were statistically different from the 16.0% for CYP2C9*2 and 10.0% for CYP2C9*3 for Spaniards and pointed to the need for optimizing dosing based on Hispanic-American allele frequency data instead of applying European or Caucasian allele frequencies to Hispanic populations.[10] The allele frequencies for our Hispanic population of 5.73% for CYP2C9*2, 3.28% for CYP2C9*3, and 46.64% for VKORC1 3673 were similar to these other studies. From what is known of these warfarin SNPs in the Hispanic population, the allele frequencies most closely align with the Caucasian population. However, it should be noted that the Hispanic population tends to have slightly lower CYP2C9*2 and higher VKORC1 3673 frequencies than the Caucasian population (Table 3). The wide inter-ethnic variability of allele frequencies for CYP2C9*2, CYP2C9*3, and VKORC1 3673 has previously been shown to explain much of the inter-ethnic variability in warfarin dosing.[8,6,12,26,28]

Finally, a recent multi-national NIH funded study published in the *New England Journal of Medicine*[34] has used data on over

4,000 patients to establish a genetics-based dosing algorithm for warfarin. This algorithm uses basic patient characteristics, such as age, gender, and ethnicity, along with the patient's known CYP2C9 and VKORC1 SNPs to more accurately predict a patient's warfarin maintenance dose than standard dosing algorithms. While the algorithm has been validated, it is not yet known if using this new pharmacogenetic-based algorithm will decrease the cost of warfarin treatment and dosing titration as well as the drug's adverse effects.

A number of small studies have indicated a decreased incidence of warfarin adverse events when genetics-based dosing algorithms are used in initiating warfarin therapy versus standard dosing algorithms.[35,36] However, a few other studies have failed to show a statistical difference in adverse events and outcomes between genetic-based and standard algorithms.[37] Larger randomized controlled studies will be needed to determine the superior algorithm and the cost-benefit analysis of using pharmacogenetic-based warfarin dosing. Also, while a few of the small randomized controlled studies that have been completed have boasted a quick turn-around time of genotyping in 1-4 hours, these studies still used research laboratories for testing.[24,35-37] Clinical laboratory and point of care testing for both the CYP2C9 and VKORC1 SNPs are being developed and will likely reach mainstream markets in a year or two. Once this occurs we are likely to see increased use of pharmacogenetic-based warfarin dosing.

CONCLUSION

Knowledge of a patient's warfarin sensitivity SNPs is likely to benefit emergency medicine by decreasing the number of inpatient admissions for warfarin titration and decreasing ED visits for warfarin adverse events. Practitioners can well imagine a clinical scenario in which a patient is low risk for VTE complications but high risk for anticoagulation complications, for example the elderly. Knowledge of warfarin sensitivity SNPs would allow the emergency physician to tailor the warfarin dose specifically for the patient, thereby decreasing the risk of elevated INR and bleeding. The patient might then be safely discharged home with outpatient follow up instead of admission. As technology for genetic testing improves, a patient's warfarin susceptibility SNPs might become part of an emergency physician's evaluation and treatment of VTE. Our pilot study shows that allele frequencies of target warfarin sensitivity SNPs are frequent enough to make personalized warfarin dosing in the ED feasible.

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New Functional Imaging Technology to Differentiate between Chronic Obstructive Pulmonary Disease and Heart Failure

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We are investigating the potential role for vibration response imaging (VRI) in the prompt emergency department (ED) evaluation of patients presenting with dyspnea to aid in differentiation between chronic obstructive pulmonary disease (COPD) and heart failure (HF). VRI is a noninvasive, bedside computer-based technology that creates a visually dynamic two-dimensional image of distribution of vibration within the lungs during the respiratory process. The acoustic signals, recorded from 36 posteriorly positioned surface skin auditory sensors, are transferred to a hardware board where several stages of filtering are applied to select a specific frequency band. The filtered output signal frequencies are presented as a gray-scale coded dynamic image consisting of a series of 0.17 s frames and as a table featuring the percentage contribution of each lung and lung sector (upper, middle, lower) to the total vibration signal. Darker images represent more prominent airway vibration, but do not necessarily differentiate laminar from turbulent flow. These images and calculations may be useful in delineating HF from other lung disease in acutely dyspneic patients.[1]

The patient whose image is presented here is a 55-year-old male who presented with shortness of breath and bilateral lower extremity edema. Vital signs at triage were pulse of 88 beats per minute, blood pressure of 160/108 mmHg and respiratory rate of 18 breaths per minute. He gave no history of respiratory or cardiac disease but was a long-time heavy tobacco smoker; his portable chest radiograph was unremarkable. Serum brain natriuretic peptide (BNP) was 136 pg/ml. At the bedside, the emergency physician obtained a VRI (Figure).

VRI clearly showed bibasilar diminution both in the image (note the darker intensity as well as broader area in the upper lungs) and as quantified in the table. This pattern in VRI is typical for an acute HF presentation. Bedside transthoracic echocardiography revealed an estimated left ventricular ejection fraction of only 35%. The patient was given intravenous diuretic and nitroglycerin therapy resulting in

Calculated for frames from 1 to 71

	Right	Left
Upper	24%	13%
Middle	21%	21%
Lower	11%	10%
TOTAL	56%	44%

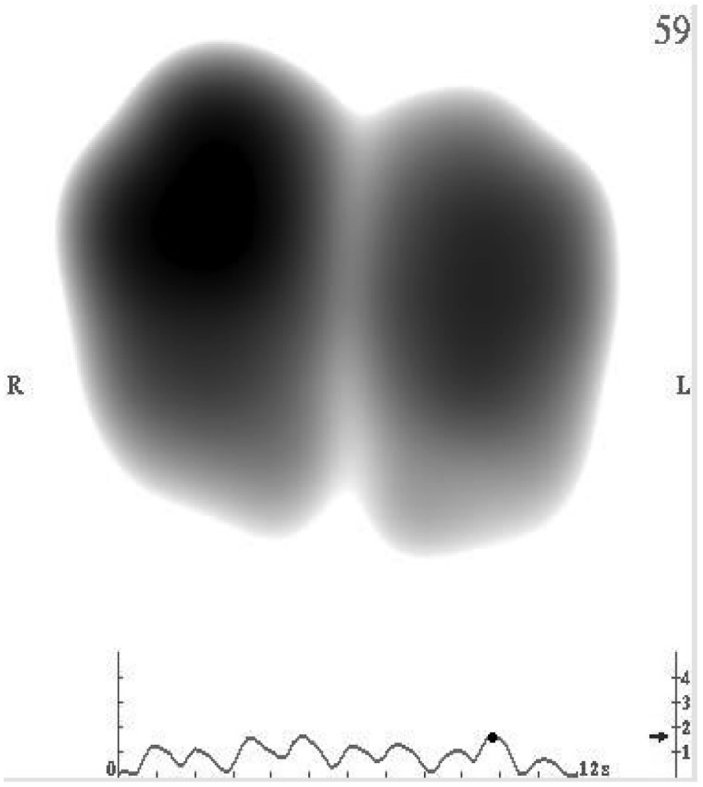

Figure. Components of vibration response imaging include visual and quantitative phonographic characterization of the lungs. In this patient there is bibasilar diminution both in the image (note the darker intensity as well as broader area in the upper lungs) and as quantified in the table.

significant improvement in his dyspnea and reduction in his blood pressure to 146/96 mmHg. As an inpatient, serial troponin assays were within normal limits and the patient responded well to therapy for heart failure. The VRI was more helpful than the BNP level in guiding acute management in the ED; the limitations of BNP have been recently discussed.[2]

VRI may be useful in the ED for differentiating the origin of acute dyspnea. An ED-based prospective trial has begun to investigate this use.

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Detailed Analysis of Prehospital Interventions in Medical Priority Dispatch System Determinants

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Background: Medical Priority Dispatch System (MPDS) is a type of Emergency Medical Dispatch (EMD) system used to prioritize 9-1-1 calls and optimize resource allocation. Dispatchers use a series of scripted questions to assign determinants to calls based on chief complaint and acuity.

Objective: We analyzed the prehospital interventions performed on patients with MPDS determinants for breathing problems, chest pain, unknown problem (man down), seizures, fainting (unconscious) and falls for transport status and interventions.

Methods: We matched all prehospital patients in complaint-based categories for breathing problems, chest pain, unknown problem (man down), seizures, fainting (unconscious) and falls from January 1, 2004, to December 31, 2006, with their prehospital record. Calls were queried for the following prehospital interventions: Basic Life Support care only, intravenous line placement only, medication given, procedures or non-transport. We defined Advanced Life Support (ALS) interventions as the administration of a medication or a procedure.

Results: Of the 77,394 MPDS calls during this period, 31,318 (40%) patients met inclusion criteria. Breathing problems made up 12.2%, chest pain 6%, unknown problem 1.4%, seizures 3%, falls 9% and unconscious/fainting 9% of the total number of MPDS calls. Patients with breathing problem had a low rate of procedures (0.7%) and cardiac arrest medications (1.6%) with 38% receiving some medication. Chest pain patients had a similar distribution; procedures (0.5%), cardiac arrest medication (1.5%) and any medication (64%). Unknown problem: procedures (1%), cardiac arrest medication (1.3%), any medication (18%). Patients with Seizures had a low rate of procedures (1.1%) and cardiac arrest medications (0.6%) with 20% receiving some medication. Fall patients had a lower rate of severe illness with more medication, mostly morphine: procedures (0.2%), cardiac arrest medication (0.2%), all medications (28%). Unconscious/fainting patients received the following interventions: procedures (0.3%), cardiac arrest medication (1.9%), all medications (32%). Few stepwise increases in the rate of procedures or medications were seen as determinants increased in acuity.

Conclusion: Among these common MPDS complaint-based categories, the rates of advanced procedures and cardiac arrest medications were low. ALS medications were common in all categories and most determinants. Multiple determinants were rarely used and did not show higher rates of interventions with increasing acuity. Many MPDS determinants are of modest use to predict ALS intervention. [West J Emerg Med. 2011;12(1):19-29.]

INTRODUCTION

Emergency Medical Dispatch (EMD) is a system of categorizing and prioritizing emergency calls in order to send an appropriate ambulance response. A variety of studies in differing systems with both health and non-health trained dispatchers have been published using a variety of different clinical measures to gauge success.[1-12] The Medical Priority Dispatch System (MPDS) is a complex computer-based EMD system that uses callers' responses to scripted questions to categorize cases into numerical complaint-based categories, which are further assigned a priority (Alpha, Bravo, Charlie, Delta, or Echo) based on their perceived acuity. Alpha and Bravo represent the lowest acuity calls, with Charlie, Delta and Echo representing higher acuity calls that may require advanced assessment and/or intervention. Calls may be further assigned a numerical subgroup and a modifier, which provide responders with more specific details about the call. Together, the complaint-based numerical category, priority (Alpha through Echo), subgroup and modifier (when present) make up the MPDS determinant. For instance, a call may be assigned to the MPDS determinant 6D2A. The number six is the complaint-based category for breathing problems, "D" (or Delta) represents priority. Two is a subcategory that informs providers that the patient is not alert, and "A" is a modifier that indicates the patient has a history of asthma.

Several studies have examined the predictive accuracy of MPDS and other EMD systems for a variety of outcomes, including paramedic-assigned acuity score, physician diagnosis of an acute illness, cardiac arrest, "Code 3" or "lights and sirens" return, and the need for Advanced Life Support (ALS) intervention.[9, 10, 13-18] Most research has demonstrated that MPDS and other EMD systems identify most but not all urgent calls with a considerable degree of overtriage.[7-10, 12, 14, 16, 19-21]

Patients with breathing problems make up approximately 11-15% of all Emergency Medical Services (EMS) calls.[9, 15] Because there are no low priority determinants related to respiratory distress, it has a sensitivity of 100% to predict ALS intervention, and the positive predictive value has ranged from 44-84%.[9, 11] Previous studies have demonstrated little difference in medication administration rates (range of 40 to 65%) and airway procedure rate (1%) between the various subgroups of patients with breathing problems.[11, 12] A recent study demonstrated increasing rates of cardiac arrest (0.1% to 17%) with increasing category severity.[15]

Patients with chest pain make up approximately 10% of EMS calls.[9] One study examined the differences between chest pain determinants and demonstrated that they all had relatively high rates of medication administrations (40% to 65%), usually aspirin or nitroglycerin.[11] Fewer than 1% of each determinant required advanced airway maneuvers, cardiac arrest medications or defibrillation.[12]

Because few (1%) of the chest pain calls were classified as Basic Life Support (BLS), the sensitivity of EMD was excellent (99-100%) to predict ALS interventions but with very poor specificity (0-2%).[9, 11] Another study of the London EMS system also demonstrated infrequent use of the alpha priority among chest pain patients and a consistently low rate of cardiac arrest among all determinant (<1%).[16] The Alpha priority had a lower rate of "lights and sirens" return (3%) as compared to Charlie and Delta priorities (both 12%).

Patients with an unknown problem (man down) make up approximately 3% of all EMS calls.[9] In a previous study in our system, this category had a sensitivity of 36% with a specificity 85% for ALS interventions.[22] One study of 696 patients classified as an unknown problem had an advanced procedure rate of 1% and medications were administered to 22%.[15] A more in-depth analysis of the unknown problem complaint-based category revealed that the rate of cardiac arrest among those with a bravo priority were all less than 1% and modestly higher (1.5%) rate of those with Delta (life status questionable).[15]

Patients with a seizure make up approximately 4% of all EMS calls.[9, 15] The MPDS determinants for seizure have been demonstrated to have a reasonable sensitivity (83%) and poor specificity (20%) to predict the need for ALS.[11] Another study from Toronto demonstrated that determinants for seizure had 66% sensitivity and a 46% specificity for Canadian Triage and Acuity Scale (CTAS) score.[9]

It has been described that approximately 3% of cardiac arrest calls are initially categorized as a seizure.[9, 14, 23, 24] The 12D1 determinant (seizing not breathing) was infrequently used in one study but had an extraordinarily high rate of cardiac arrest (26%).[14] Those patients with a known history of seizure had a clinically insignificant rate of cardiac arrest.

Patients with a history of falls make up 11% of all EMS calls.[9] One study examined 103 calls from the falls complaint-based category and demonstrated that 26% of these were found to have important clinical field findings and that these had a modest correlation with age.[25] The rates of ALS interventions given to fall patients in the lowest priority (Alpha) determinants ranged from 7-10% in one system and 46% in another.[3, 5, 6] The rate of ALS interventions for all Fall patients in another study was 27%.[26] That same Toronto study demonstrated that the fall category had a 20% sensitivity and a 93% specificity for CTAS score.[9]

Patients in the fainting/unconscious category make up 9% of all EMS calls.[9] The MPDS determinants for fainting/unconscious have been demonstrated to have good sensitivity (92%) and very poor specificity (2%) to predict the need for ALS.[11] The Toronto study demonstrated that the fainting/unconscious category had a 98% sensitivity and a 9% specificity for CTAS score.[9]

This descriptive analysis of an individual EMS system is the first to provide a comprehensive evaluation of interventions performed in the specific MPDS determinants for these common problems for both transported and non-transported patients. This study asks the following question:

do the multiple determinants, subgroups, and modifiers in the breathing problems, chest pain, unknown problem (commonly known as "man down"), seizure, falls and fainting/unconscious categories aid in predicting prehospital interventions?

METHODS

San Mateo County is an urban/suburban county of 552 square miles with a population of 700,000. It receives approximately 40,000 calls for emergency medical assistance annually. All calls receive an ALS response under a tiered system, consisting of a fire department single paramedic first response team and a private ambulance staffed with at least one paramedic. An electronic prehospital care record is established for each patient, which includes patient demographics, medical history, signs and symptoms, and clinical interventions.

Nine-one-one callers are asked a series of scripted questions that include the patient's level of consciousness, age, chief complaint and other complaint-specific questions. A computer-aided dispatch system records general information regarding each call, including date, time, location of call, dispatch time, dispatch code and disposition. We used MPDS (Versions 11.2 and 11.3 (May 2006), NAEMD, Salt Lake City, UT) to categorize cases into standardized, complaint-based categories, which were further classified as Alpha or Bravo for no "lights and sirens" response, and Charlie, Delta, or Echo for "lights and sirens" response. In some cases, a subgroup or modifier was added to alert providers to further details about the call.

All EMS patients from January 1, 2004, to December 1, 2006, were identified from the Computer Aided Dispatch system and linked automatically by call number to an electronic prehospital care record. We electronically imported data into a Excel spreadsheet (Microsoft Corporation, Redmond, WA). In this study we included all patients assigned a priority by MPDS in the categories of breathing problems, chest pain, unknown problem (commonly known as "man down"), seizure, falls and fainting/unconscious. The investigators chose these complaint-based categories a priori, as they make up a significant portion of EMS calls and encompass a large number of prehospital interventions. Furthermore, several of these categories have had mixed results or inconclusive results about their predictive abilities for prehospital interventions in prior studies. The University of California, San Francisco Committee on Human Research approved this study.

ALS level of care was defined as those patients who received a procedure, a medication or an intravenous (IV) fluid infusion. Medications available in the San Mateo County EMS system included nitroglycerin, aspirin, adenosine, albuterol, atropine, epinephrine, dopamine, diphenhydramine, lidocaine, naloxone, glucagon, midazolam, sodium bicarbonate, dextrose 50%, morphine, activated charcoal, oral glucose, glucose cola and intravenous fluid. Oxygen was not included as a medication. Intravenous fluid was defined either as an infusion of a volume greater than 100cc or as a chart in which the phrases "wide open" or "infusion" were noted. Procedures included endotracheal intubation, Combitube placement, defibrillation, transcutaneous pacing, cardioversion, needle cricothyrotomy or needle thoracotomy. Blood glucose measurement, wound care, splinting, cervical spine immobilization and pulse oximetry were not included as procedures, as these are considered BLS skills in our system. We also excluded IV catheter placement without fluid infusion, as prior studies in our system demonstrated a high rate of intravenous line insertion in low acuity patients.[6] Patients who did not receive a medication or a procedure were considered to have received BLS level of care. Patients not transported to the hospital were placed in a non-transport category.

We analyzed calls in the complaint-based categories for breathing problems, chest pain, unknown problem (man down) seizures, fainting (unconscious) and falls for transport status and ALS interventions. They were queried for the following prehospital interventions: BLS care only, intravenous line placement only, medication given, procedures or non-transport. We tabulated the numbers and percentages of each of these interventions tabulated for each determinant. We directly compared percentages of prehospital interventions in each category, and assessed statistical significance via a two-tailed paired t-test using Statistics Calculator (StatPac Inc., Bloomington, MN).

RESULTS

A total of 77,394 calls underwent the EMD process during the study period. The number and percentage of the total patients in each category are as follows: breathing problems 9,435 (12.2%), chest pain 4,679, (6.0%), unknown problem 1,094 (1.4%), seizure 2,606 (3.4%), falls 6,741 (8.7%), and fainting/unconscious 6,763 (8.7 %).

Among those patients with breathing problems, the far majority of the patients were classified as 6D1, severe respiratory distress (Figure 1). There was no significant difference in the rate of advanced procedures between those with 6D1 (1.2%) and 6D1A (with asthma) [0.9% p=0.33].

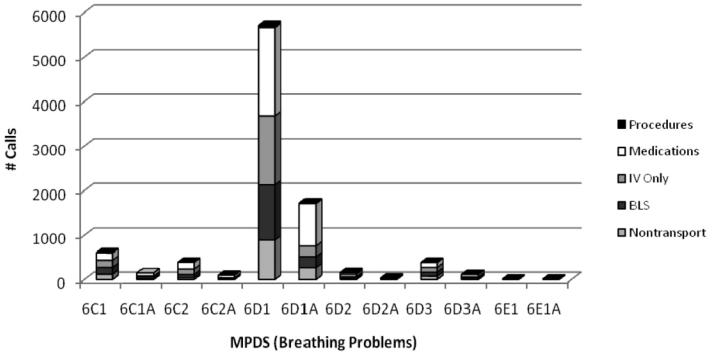

Figure 1. Breathing problem patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

Table 1. Procedures by Medical Priority Dispatch System (MPDS) determinant.

Description	MPDS	Number of calls	Advanced airway	Defibrillation	Cardioversion	Cardiac pacing	Cricothyrotomy
Breathing problems	**6Total**	**9435**	**66**	**9**	**8**	**5**	**0**
Abnormal breathing	6C1	616	1	0	0	0	0
Abnormal breathing+*asthma*	6C1A	153	0	0	0	0	0
Cardiac history	6C2	394	0	0	0	0	0
Cardiac history+*asthma*	6C2A	98	0	0	0	0	0
Severe respiratory distress	6D1	5719	50	7	8	3	0
Severe respiratory distress+*asthma*	6D1A	1723	14	0	0	2	0
Not alert	6D2	164	0	1	0	0	0
Not alert+*asthma*	6D2A	35	0	0	0	0	0
Clammy	6D3	393	1	0	0	0	0
Clammy+*asthma*	6D3A	114	0	1	0	0	0
Ineffective breathing	6E1	13	0	0	0	0	0
Ineffective breathing+*asthma*	6E1A	13	0	0	0	0	0
Chest pain (non-traumatic)	**10Total**	**4679**	**7**	**9**	**10**	**6**	**1**
Breathing normally	10A1	56	0	0	0	0	0
Abnormal breathing	10C1	825	1	0	0	1	0
Cardiac history	10C2	667	0	0	1	0	0
Cocaine	10C3	2	0	0	0	0	0
Breathing normally > 35	10C4	651	0	0	0	0	0
Severe respiratory distress	10D1	1263	1	1	2	4	0
Not alert	10D2	215	1	1	0	1	1
Clammy	10D3	1000	4	7	7	0	0
Unknown problem (man down)	**32Total**	**1094**	**11**	**17**	**0**	**1**	**0**
Standing, sitting, moving, or talking	32B1	390	4	0	0	0	0
Medical alert notifications	32B2	415	0	0	0	0	0
Unknown status (3rd party caller)	32B3	124	3	6	0	0	0
Life status questionable	32D1	165	4	11	0	1	0
ALL CATEGORIES	**TOTAL:**	**15208**	**84**	**35**	**18**	**12**	**1**

There was a small but statistically significant difference in the advanced procedure rate of 6C determinants (0%), as compared to 6D (1.1% $p<0.01$) [Table 1]. Less than 1% of all patients with breathing problems received a procedure. Of the 88 patients with breathing problems who received a procedure, most received an advanced airway.

Albuterol and nitroglycerin were the most common medications among those patients with a breathing problem with 38% of all these patients receiving some medication. One hundred forty-nine of these patients (1.6%) received cardiac arrest drugs (Table 2).

For those patients with chest pain there was a wide distribution of interventions among determinants, except for the rarely used 10A1 determiant (Figure 2). There was a small stepwise increase in the rate of procedures or medications seen in this category, with 53% of calls in the 10C determinant receiving procedures or medications compared with 49% in 10D. ($p=0.02$) [Tables 1 and 2]. Only 23 (0.5%) of the patients with chest pain received a procedure, with some of these patients receiving multiple procedures (Table 1). The

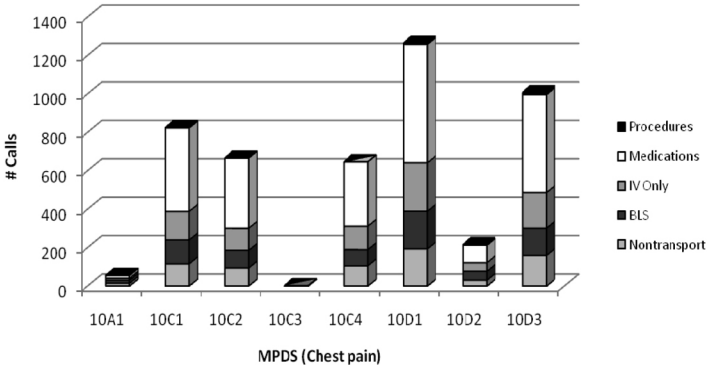

Figure 2. Chest pain patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

Table 2. Medications by Medical Priority Dispatch System (MPDS) determinants.

Description	MPDS	Number of calls	Nitroglycerin	Aspirin	Midazolam	Naloxone	Glucose	Cardiac arrest medications	Albuterol	Morphine	Intravenous infusion
Breathing problems	**6Total**	**9435**	**2010**	**409**	**38**	**38**	**169**	**149**	**4228**	**170**	**389**
Abnormal breathing	6C1	616	70	17	0	0	6	5	183	11	22
Abnormal breathing+*asthma*	6C1A	153	17	6	0	0	3	1	108	0	0
Cardiac history	6C2	394	129	26	3	1	3	1	164	9	5
Cardiac history +*asthma*	6C2A	98	23	8	0	0	1	1	76	2	3
Severe respiratory distress	6D1	5719	1315	261	22	26	132	86	1953	107	286
Severe respiratory distress+*asthma*	6D1A	1723	322	64	12	6	13	45	1500	22	46
Not alert	6D2	164	9	4	0	5	4	3	18	0	14
Not alert+*asthma*	6D2A	35	3	3	0	0	1	1	13	0	0
Clammy	6D3	393	102	17	1	0	6	1	102	19	12
Clammy+*asthma*	6D3A	114	20	3	0	0	0	1	86	0	1
Ineffective breathing	6E1	13	0	0	0	0	0	0	0	0	0
Ineffective breathing +*asthma*	6E1A	13	0	0	0	0	0	4	25	0	0
Chest pain (non-traumatic)	**10Total**	**4679**	**3678**	**1835**	**14**	**4**	**33**	**72**	**243**	**221**	**133**
Breathing normally	10A1	56	21	13	0	1	0	0	0	0	0
Abnormal breathing	10C1	825	731	327	1	0	2	8	97	36	9
Cardiac history	10C2	667	548	295	1	0	3	2	11	41	15
Cocaine	10C3	2	0	0	0	0	0	0	0	0	0
Breathing normally > 35	10C4	651	510	277	0	0	2	6	5	20	15
Severe respiratory distress	10D1	1263	904	457	7	1	20	24	84	69	50
Not alert	10D2	215	118	64	2	2	1	5	6	8	19
Clammy	10D3	1000	846	402	3	0	5	27	40	47	25
Unknown problem (man down)	**32Total**	**1094**	**98**	**36**	**4**	**20**	**34**	**13**	**54**	**58**	**57**
Standing, sitting, moving, or talking	32B1	390	10	8	2	6	14	5	9	17	17
Medical alert notifications	32B2	415	80	25	0	1	10	1	40	34	14
Unknown status (3rd party caller)	32B3	124	2	1	1	5	5	2	4	7	14
Life status questionable	32D1	165	6	2	1	8	5	5	1	0	12
All Categories	**TOTAL:**	**15208**	**5786**	**2280**	**56**	**62**	**236**	**234**	**4525**	**449**	**579**

procedures in this category were distributed between advanced airways (7), defibrillation (9), cardioversion, (10) transcutaneous pacing (6) and cricothyrotomy (1). Most of these procedures occurred in the Delta and Echo priorities. Of the 4,679 patients with chest pain, 79% received nitroglycerin and 39% received aspirin (Table 2). Seventy-two (1.5%) patients with chest pain received cardiac arrest drugs.

For those patients in the unknown problem (man down) category, there was no increase in medication or procedure rates for higher priority calls (Figure 3). The incidence of procedure and medication use for 32B determinants was 19% compared with 17% in 32D (p=0.56). Among patients with an unknown problem, 1% received a procedure, mostly accounted for by airway and defibrillation (Table 2).

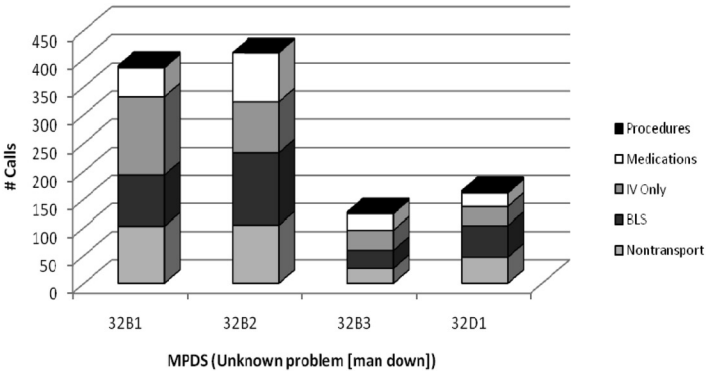

Figure 3. Unknown problem patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

Approximately 18% of patients received a medication and 1.3% received cardiac arrest medications in this category (Table 2).

The majority (84%) of the seizure calls fell into three determinants: 12A1, 12D2, or 12D3 (Figure 4). Fifteen (0.6%) patients among the seizure calls received cardiac arrest medications with no discernable pattern among determinants (Table 3). One determinant (12D1, seizure and not breathing) had two out of seven (29%) cases requiring cardiac arrest medications, which was markedly higher than others in this category.

An examination of the rate of procedures among seizure patients produced no discernible pattern (Figure 4 and Table 4). The most commonly administered medications were midazolam and glucose (Table 3). The rate of midazolam administration demonstrated a higher rate among the Delta subcategories (Alpha 4%, Bravo 3%, Charlie 3%, Delta 13%, p<0.02). The subcategory of 12A1 (seizing stopped and breathing regularly) had a 5% rate of midazolam administration. Those patients with continuous or multiple seizures (12D2, 12D2E) had a 17% rate of midazolam administration with a known history of epilepsy making no significant difference.

For those patients with the complaint of falls, there was a wide distribution of interventions among determinants (Figure 5). The rate of advanced airway and cardiac arrest medication among the fall group was very low (0.2%) [Table 3]. Morphine, the most common medication received by fall patients, was administered to 24% of the total. There was no obvious pattern to the rate of morphine administration among the subgroups. The Omega subcategory (17O1 public assist with no injuries or priority symptoms) was rarely used but had no procedures or morphine administration.

Among those patients in the fainting/unconscious category, there was an increasing rate of medication administration with higher priority (Alpha 15%, Charlie, 25%, Delta 36%, p-value for trend <0.01) [Table 3]. There was no consistent pattern in the types of medications given to these patients (Table 3). The rate of cardiac arrest medication

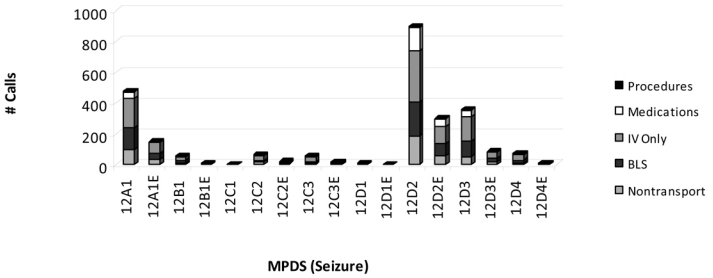

Figure 4. Seizure patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

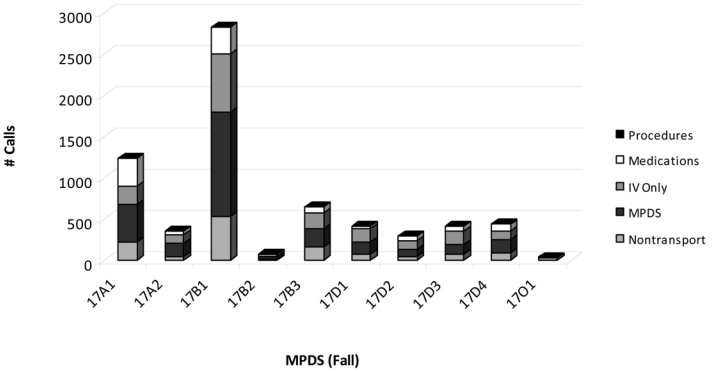

Figure 5. Fall patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

administration increased with higher priority (Alpha 0%, Charlie, 1%, Delta 2.6%, p-value comparing Charlie to Delta <0.01). The rates of advanced airway intervention among the Fainting/Unconscious determinants were all well below 1%, except for the still unconscious (31D1) determinant (3.7%).

DISCUSSION

The MPDSand other EMD systems are designed to aid in the decision to send which prehospital resource and at what level of urgency. The use of multiple determinants and subgroups in each complaint category should aid in these decisions by having demonstrably increasing rates of ALS procedures and medication administration with higher priority.

These six MPDS complaint-based categories collectively accounted for 40% of calls that underwent the EMD process. The procedure rate was low (<1%) in four categories (breathing problem, chest pain, unknown problem and falls) and was only slighter higher in two others (seizure 1.1%, and fainting/unconscious 2.9%).

The rate of administration of cardiac arrest drugs was low (<1%) for most of the categories with the exception of ineffective breathing (31% cardiac arrest), chest pain clammy (3%), man down life status questionable (3%), seizure not breathing (28%) and unconscious with severe respiratory distress (5%). The rate of cardiac arrests among the seizure patients (0.6%) was similar to other studies.[17] The high rate of cardiac arrest among those seizure patients classified as 12D1

Table 3. Medications by Medical Priority Dispatch System (MPDS) determinants.

Description	MPDS	Number of calls	Nitroglycerin	Aspirin	Midazolam	Naloxone	Glucose	Cardiac arrest medications	Albuterol	Morphine	Intravenous infusion
Convulsions/ Seizures total	**12Total**	**2606**	**19**	**13**	**259**	**23**	**113**	**15**	**15**	**0**	**62**
Not seizing now and breathing regularly	12A1	479	3	3	24	5	11	2	3	0	17
Not seizing now and breathing regularly *+Epilepsy*	12A1E	152	0	0	3	0	3	0	1	0	0
Breathing regularly not verified <35	12B1	60	0	0	2	0	2	0	0	0	5
Breathing regularly not verified <35+*Epilepsy*	12B1E	10	0	0	0	0	1	0	0	0	0
Pregnancy	12C1	3	0	0	0	0	0	0	0	0	0
Diabetic	12C2	70	2	0	2	1	12	0	3	0	1
Diabetic+*Epilepsy*	12C2E	22	0	0	0	0	2	0	0	0	1
Cardiac history	12C3	56	1	1	0	1	1	1	0	0	5
Cardiac history+ *Epilepsy*	12C3E	21	0	0	3	0	1	0	0	0	0
Not breathing	12D1	7	0	0	0	0	0	2	0	0	1
Not breathing+ *Epilepsy*	12D1E	2	0	0	0	0	0	0	0	0	0
Continuous or multiple seizures	12D2	895	7	4	149	10	44	4	3	0	19
Continuous or multiple seizures+*Epilepsy*	12D2E	301	2	1	48	0	18	0	3	0	6
Irregular breathing	12D3	354	4	4	17	6	12	4	1	0	4
Irregular breathing+ *Epilepsy*	12D3E	86	0	0	2	0	2	1	0	0	0
Breathing regularly not verified > 35	12D4	76	0	0	7	0	4	1	1	0	3
Breathing regularly not verified >35+*Epilepsy*	12D4E	12	0	0	2	0	0	0	0	0	0

(seizure and not breathing, 29%) was similar to a prior study, although with small numbers in both studies.[14]

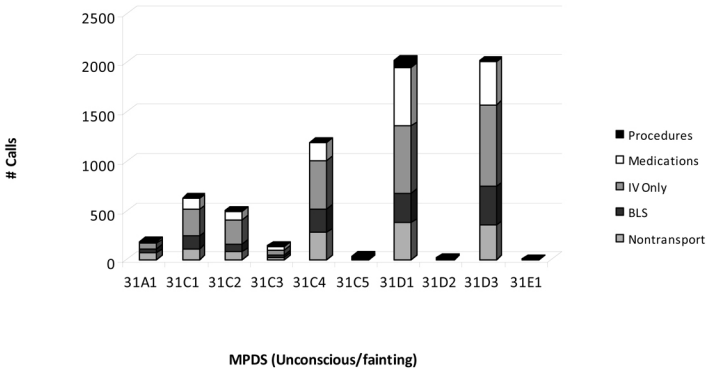

Figure 6. Unconscious/fainting patients by type of intervention. *MPDS,* Medical Priority Dispatch System; *IV,* intravenous; *BLS,* basic life support

The overall medication rate for each category was seizure 20%, falls 28% and fainting/ unconscious 32%. Those patients from the seizure category were most often treated with a benzodiazepine and glucose. Among fall patients, morphine was the most common medication given but accounted for only 24% of the total. Fainting/unconsciousness patients were treated with a broad range of medications.

Prior studies have demonstrated that the addition of ALS resources to a prehospital system causes a modest decrease in mortality among patients with breathing problems or chest pain.[27, 28] It is likely that some of these prehospital treatments are time dependent, but it is not known whether a "lights and sirens" response is required to achieve these improvements.

Among those patients with breathing problems, the far majority were classified as severe respiratory distress (6D1), similar to an earlier study.[15] We saw little difference in the

Table 3 (continued)

Description	MPDS	Number of calls	Nitroglycerin	Aspirin	Midazolam	Naloxone	Glucose	Cardiac arrest medications	Albuterol	Morphine	Intravenous infusion
Falls total	**17Total**	**6741**	**17**	**17**	**5**	**20**	**58**	**13**	**49**	**1589**	**120**
Not dangerous body area	17A1	1233	1	2	0	0	4	1	3	650	13
Non-recent (>6h) injuries (no priority symptoms)	17A2	348	0	0	0	2	0	0	3	56	5
Possibly dangerous body area	17B1	2826	7	10	0	1	17	4	6	523	28
Serious hemorrhage	17B2	79	0	0	0	0	0	0	0	11	2
Unknown status (3rd party caller)	17B3	658	2	2	0	3	12	1	3	96	14
Dangerous body area	17D1	413	2	2	1	5	3	1	4	11	15
Long fall (>6 feet/2 meters)	17D2	296	0	0	2	2	0	0	0	128	4
Unconscious or not alert	17D3	415	0	0	1	7	20	3	4	23	24
Abnormal breathing	17D4	435	5	1	1	0	2	3	23	91	14
Public assist (no injuries; no priority symptoms)	17O1	38	0	0	0	0	0	0	3	0	1
Unconscious/ fainting (near) total	**31Total**	**6763**	**222**	**154**	**39**	**249**	**291**	**128**	**208**	**50**	**801**
Single or near fainting episode and alert <35	31A1	193	0	0	1	0	7	0	1	0	20
Alert with abnormal breathing	31C1	632	46	28	2	0	8	9	23	12	58
Cardiac history	31C2	503	33	34	1	1	4	8	9	9	46
Multiple fainting episodes	31C3	138	6	4	1	0	1	3	0	5	30
Single or near fainting episode and alert >35	31C4	1205	30	27	0	4	19	4	3	14	145
Females 12-50 with abdominal pain	31C5	37	0	0	0	0	0	0	0	2	7
Unconscious	31D1	1984	36	24	25	201	160	77	108	1	226
Severe respiratory distress	31D2	18	2	0	0	0	1	1	11	0	2
Not alert	31D3	2046	69	37	9	43	91	26	53	7	267
Ineffective breathing	31E1	7	0	0	0	0	0	0	0	0	0
All Categories	**TOTAL:**	**16110**	**258**	**184**	**303**	**292**	**462**	**156**	**272**	**1639**	**983**

treatment rates of those with and without asthma. This study demonstrates that most subgroups of breathing problem were rarely used and there is little difference in the rate of medications or procedure rate.[10] Due to the relatively high rate of medication administration, all patient who are assigned into breathing problem determinants in a tiered prehospital system should be sent with an ALS response. It is less clear whether lights and sirens are required in all categories.

Among patients with chest pain, very few were placed in the 10A1 determinant. Those infrequent chest pain patients who received a procedure or cardiac arrest medications were found in almost all subgroups.The administration of other medications was common in all determinants (most commonly aspirin and nitroglycerin) making these distinctions to be of questionable use in refining an EMS response. Again, due to the relatively high rate of medication administration, patients

Table 4. Procedures by Medical Priority Dispatch System (MPDS) determinant.

Description	MPDS	Number of calls	Advanced airway	Defibrillation	Cardioversion	Cardiac pacing	Cricothyrotomy
Convulsions/Seizures total	**12Total**	**2606**	**8**	**18**	**2**	**1**	**0**
Not seizing now and breathing regularly	12A1	479	1	2	0	0	0
Not seizing now and breathing regularly +*Epilepsy*	12A1E	152	0	0	0	0	0
Breathing regularly not verified <35	12B1	60	0	0	0	0	0
Breathing regularly not verified <35+*Epilepsy*	12B1E	10	0	0	0	0	0
Pregnancy	12C1	3	0	0	0	0	0
Diabetic	12C2	70	0	0	0	0	0
Diabetic+*Epilepsy*	12C2E	22	0	0	0	0	0
Cardiac history	12C3	56	0	0	0	0	0
Cardiac history+*Epilepsy*	12C3E	21	0	0	0	0	0
Not breathing	12D1	7	2	8	0	0	0
Not breathing+*Epilepsy*	12D1E	2	0	0	0	0	0
Continuous or multiple seizures	12D2	895	3	4	0	1	0
Continuous or multiple seizures +*Epilepsy*	12D2E	301	0	0	0	0	0
Irregular breathing	12D3	354	2	3	0	0	0
Irregular breathing+*Epilepsy*	12D3E	86	0	0	0	0	0
Breathing regularly not verified >35	12D4	76	0	1	2	0	0
Breathing regularly not verified >35+*Epilepsy*	12D4E	12	0	0	0	0	0
Falls total	**17Total**	**6741**	**10**	**3**	**0**	**2**	**0**
Not dangerous body area	17A1	1233	0	0	0	0	0
Non-recent (>6h) injuries (no priority symptoms)	17A2	348	0	0	0	0	0
Possibly dangerous body area	17B1	2826	1	0	0	0	0
Serious hemorrhage	17B2	79	0	0	0	0	0
Unknown status (3rd party caller)	17B3	658	0	0	0	0	0
Dangerous body area	17D1	413	1	0	0	1	0
Long fall (>6 feet/2 meters)	17D2	296	1	0	0	0	0
Unconsious or not alert	17D3	415	5	3	0	0	0
Abnormal breathing	17D4	435	2	0	0	1	0
Public assist (no injuries; no priority symptoms)	17O1	38	0	0	0	0	0
Unconscious/fainting (near) total	**31Total**	**6763**	**85**	**97**	**3**	**10**	**0**
Single or near fainting episode and alert <35	31A1	193	0	0	0	0	0
Alert with abnormal breathing	31C1	632	1	0	0	3	0
Cardiac history	31C2	503	1	1	0	2	0
Multiple fainting episodes	31C3	138	0	0	0	1	0
Single or near fainting episode and alert >35	31C4	1205	0	0	0	0	0
Females 12-50 with abdominal pain	31C5	37	0	0	0	0	0
Unconscious	31D1	1984	73	88	0	4	0
Severe respiratory distress	31D2	18	0	0	0	0	0
Not alert	31D3	2046	10	8	3	0	0
Ineffective breathing	31E1	7	0	0	0	0	0
All Categories	**TOTAL:**	**16110**	**103**	**118**	**5**	**13**	**0**

who are placed in the chest pain category in a tiered prehospital system should be sent with an ALS response unless BLS providers are capable of administering medications such as aspirin and nitroglycerin.

In our study there are numerous determinants that are rarely used, particularly in the seizure category. The medication rate was less than 5% in the lower acuity seizure determinants and 13% among the Delta priorities. The need for infrequent medication must be balanced against the over triage rate.

Patients in the fall category had a consistently small rate of cardiac arrest and procedures but a high rate of medication use. The types of medications administered are less time dependent (i.e. pain medications) and may allow for a lower priority response for most of these determinants.

Those patients in the fainting/unconscious category had an increasing rate of cardiac arrest medication with higher priority. These were 1% or lower in the Alpha and Charlie priorities and 2.6% in the Delta group. This category worked reasonably well at differentiating those patients who required a rapid EMS response.

Those patients in the unknown problem (man down) determinants had a similar rate of procedures (1%) and cardiac arrest medication administration (1.4%). The medication list for this category was more varied with no discernable pattern. The appropriate response for this category is less clear.

This study used process measures (procedures and medication administration) as proxies for requiring ALS intervention. This determination of the appropriate threshold of ALS dispatch must take into account the local tolerance for the rate of missed ALS calls and the cost and system implications of overtriage. An accepted hierarchy of time dependent interventions and thresholds for under-triage are necessary for the judicious analysis and optimal design of a tiered EMS system.[22] This study also is indicative of the inherent difficulty in getting adequate information from 9-1-1 callers that will allow us to make subtle clinical distinctions.

The MPDS has multiple advantages, including its computerization, the consistency of the education and usage, as well as its quality improvement process. Prior studies have demonstrated its ability to improve the diagnosis of cardiac arrest.[2] This study demonstrates that the multiple determinants in the categories of breathing problem, chest pain and unknown problem were of modest use in defining need for ALS procedures or medications. The categories of seizure, falls and fainting/ unconscious had consistently low rates of cardiac arrest and medical procedures.

LIMITATIONS

This study is limited by the fact that all of its data comes from one community. Another major limitation is the fact that all of our calls receive an ALS response, which may lead to higher delivery of ALS measures. The findings in our single-tiered EMS system may thus differ from those derived in multi-tiered EMS systems. This study was unable to measure protocol compliance with the use of ALS interventions or outcomes and this may not necessarily imply the need for these interventions.

Approximately 12% of EMD and transported calls were unmatched and excluded, potentially introducing a selection bias. This commonly occurred because of a mismatch between the dispatch-generated run number and the number entered by the paramedic. A large percentage of our calls (28%) were not subject to the EMD process and this also may have had an effect on data analysis. Most of these were calls for assistance by law enforcement and fire personnel and have similar rates of interventions. Two versions of the MPDS (Versions 11.2 and 11.3 (adopted May 2006)) were used during this study. Non-transported patients who may have received ALS interventions, such as albuterol or dextrose, would be categorized in the non-transport group.

CONCLUSION

The procedure rate and cardiac arrest rate was low among these common EMD categories. ALS medications were common in all categories and most determinants. Many determinants and subgroups are of questionable use. There are some trends toward more cardiac arrests and procedures among higher priority determinants. Despite these trends, there is still significant over triage and concerning rates of ALS interventions in lower acuity determinants. Other systems might consider a similar analysis as a way of determining the appropriate ambulance response for common complaints.

ACKNOWLEDGMENTS

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KAS receives compensation for medical direction from American Health and Safety Training, Inc. and the San Francisco Fire Department.

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Characteristics of Patients with an Abnormal Glasgow Coma Scale Score in the Prehospital Setting

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Objective: This cross-sectional study describes the characteristics of patients with an abnormal Glasgow Coma Scale (GCS) in the prehospital setting.

Methods: We reviewed existing prehospital care reports (PCRs) in the San Mateo County, California, emergency medical services (EMS) database from January 1 to December 31, 2007. Adults age 18 or greater with a documented GCS fit inclusion criteria. We excluded single and multisystem trauma patients, as well as patients in cardiac arrest, respiratory arrest, or listed as deceased from the study. We classified the remaining patients as a normal GCS of 15 or abnormal (defined as less than 15 at any time during paramedic contact), and then further sub-classified into mild (GCS 13-14), moderate (GCS 9-12) or severe (GCS 3-8).

Results: Of the 12,235 unique prehospital care record in the database, 9,044 (73.9%) met inclusion criteria, comprised of 2,404 (26.6%) abnormal GCS patients and 6,640 (73.4%) normal GCS patients. In the abnormal GCS category, we classified 1,361 (56.6%) patients as mild, 628 (26.1%) as moderate, and 415 (17.3%) as severe. Where sex was recorded, we identified 1,214 (50.5%) abnormal GCS patients and 2,904 (43.7%) normal GCS patients as male. Mean age was 65.6 years in the abnormal GCS group and 61.4 in the normal GCS group ($p<0.0001$). Abnormal GCS patients were more likely to have a history of conditions known to be associated, such as alcohol abuse (odds ratio [OR] 2.3, 95% confidence interval [CI]=2.75-3.00), diabetes (OR 1.34, 95% CI=1.17-1.54), substance abuse (OR 1.6, CI=1.09-2.3), stroke/transient ischemic attack (OR 2.0, CI=1.64-2.5), and seizures (OR 3.0, CI=1.64-2.5). Paramedics established intravenous (IV) access on 1,821 (75.7%, OR 1.94, CI=1.74-2.2) abnormal GCS patients and administered medications to 777 (32.3%, OR 1.01, CI=0.92-1.12). Compared to patients with normal GCS, patients with a mildly abnormal GCS were less likely to receive medications (OR 0.61, CI=0.53-0.70) while those with a moderately or severely abnormal GCS were more likely (OR 1.27, CI=1.07-1.50 and OR 2.86, CI=2.34-3.49, respectively). Of the normal GCS patients, 4,097 (61.7%) received an IV and 2,125 (32.0%) received medications by any route.

Conclusion: Twenty-seven percent of all prehospital patients in our study presented with an abnormal GCS. Prehospital patients with an abnormal GCS are more likely to be male, slightly older, and have higher rates of history of alcohol use or seizure. This group of patients had a higher rate of IV placement. Patients with a mildly abnormal GCS were less likely to receive medications while those with a moderately or severely abnormal GCS were more likely. [West J Emerg Med. 2011;12(1):30-36.]

INTRODUCTION

Altered level of consciousness (ALOC) is a commonly encountered prehospital syndrome associated with various disorders, and accounts for 4-10% of all presentations to the emergency department (ED). It is one of the most common reasons for hospital admission of patients brought to the ED by emergency medical services (EMS).[1,2] Common causes of ALOC include alcohol intoxication, hypoglycemia, illicit drugs, post-ictal states and trauma.[3]

Both physicians and paramedics may find evaluation

of a patient with ALOC challenging. The reasons include lack of knowledge of a patient's medical history, baseline mental status, and precipitating events, as well as the range of changes in mental status, from agitation to coma. Paramedics make a presumptive diagnosis, or "field impression," based on the patient's clinical presentation and on limited diagnostic tests. Interventions following standard medical protocols are guided by the provider's judgment. Interventions for patients presenting with ALOC focus on treating the presumed underlying cause and include blood glucose determination, naloxone administration, benzodiazepine administration and airway management as needed. For complicated patient presentations, paramedics may be advised or required to consult a base physician.

The complicated nature of ALOC presentations, coupled with limited understanding of this group of patients and limited diagnostic tools, presents a particular challenge for paramedics. While the history and physical exam are of paramount importance in determining the etiology of ALOC, paramedics lack training in more sophisticated assessment techniques, such as a complete cranial nerve exam or mini-mental status exam that can provide etiologic clues. Additionally, paramedics lack the benefit of imaging, laboratory studies, or invasive diagnostic procedures to determine etiology and hence rely almost entirely on history and physical exam.

Paramedic protocol standards include methods for assessing level of consciousness, such as the Glasgow Coma Scale (GCS).

Given these limitations, we studied whether prehospital patients with an abnormal GCS could provide clues to a likely etiology. We hypothesized that patients with an abnormal GCS would be fundamentally different than patients with a normal GCS in terms of demographics, medical history, and use of advanced life support (ALS) interventions, such as intravenous (IV) lines or medication administration. Further, we predicted that an abnormal GCS would be associated with a known medical condition. We hope that a better understanding of this patient population will lead to prospective studies to elucidate the optimal approach to treatment.

METHODS

San Mateo County is an urban/suburban county with 700,000 people spread over 552 square miles. San Mateo receives approximately 40,000 calls for emergency medical assistance annually. Firefighter paramedics on fire engines and American Medical Response (AMR) ambulances respond to 9-1-1 emergency medical calls. The system is a public/private partnership between AMR, the fire service agencies in San Mateo County, and the County Health Services Department's EMS office. AMR contracts with a Joint Powers Authority, made up of 17 fire service agencies, to provide paramedic first responder services.[4] All ambulances are staffed by at least one emergency medical technician-paramedic, and thus all patients receive ALS level evaluation.

Table 1. Characteristics of patient by group.

	GCS 15 (n=6640)	GCS 3-14 (n=2404)
Mean age (years)*	61.4 ±20.8	65.6±21.5
Sex (%)*		
Female	55.1	49.0
Male	43.7	50.5
Race (%)		
White	43.9	46.8
Black/African America	7.6	7.2
Hispanic/Latino	8.1	9.2
Native American/Alaskan	<0.1	0
Asian/Pacific Islander	8.1	8.8
Other/unknown*	32.3	28.0
History (%)		
Alcohol*	1.8	4.0
Diabetes*	11.1	14.5
Psychiatric	3.5	3.5
Seizure*	2.4	6.9
Stroke/TIA*	3.4	6.6
Substance use*	1.1	1.7

**Indicates statistically significant result, (p<0.05)*
GCS, Glasgow Coma Scale; *TIA,* transient ischemic attack

Each patient receiving medical attention generates an electronic prehospital care record (PCR). This record includes data regarding patient demographics, home address, medical history, signs and symptoms, clinical interventions and paramedic field impression. This cross-sectional study examined medical records of the prehospital patients in San Mateo County from January 1, 2007, to December 31, 2007. We recorded these patient encounters in a Structured Query Language searchable database with over 200 data elements per patient encounter.

Inclusion criteria were age 18 years or greater with a documented GCS and documented evaluation by San Mateo County paramedics. Exclusion criteria were cardiac or respiratory arrest, death or trauma (single or multi-system) as the primary paramedic impression. We divided patients into two groups: those with a GCS of less than or equal to 14 at any time during paramedic contact, defined as having an abnormal GCS, and those with a GCS of 15, defined as normal. For purposes of analysis, we further subdivided the abnormal GCS group into mild (GCS 13-14), moderate (GCS 9-12) and severely altered (GCS less than 8) categories to look for associations and trends between severity of ALOC and various predictors. This classification scheme is common to other studies involving abnormal GCS.[5]

The GCS has three components: eye, verbal and motor

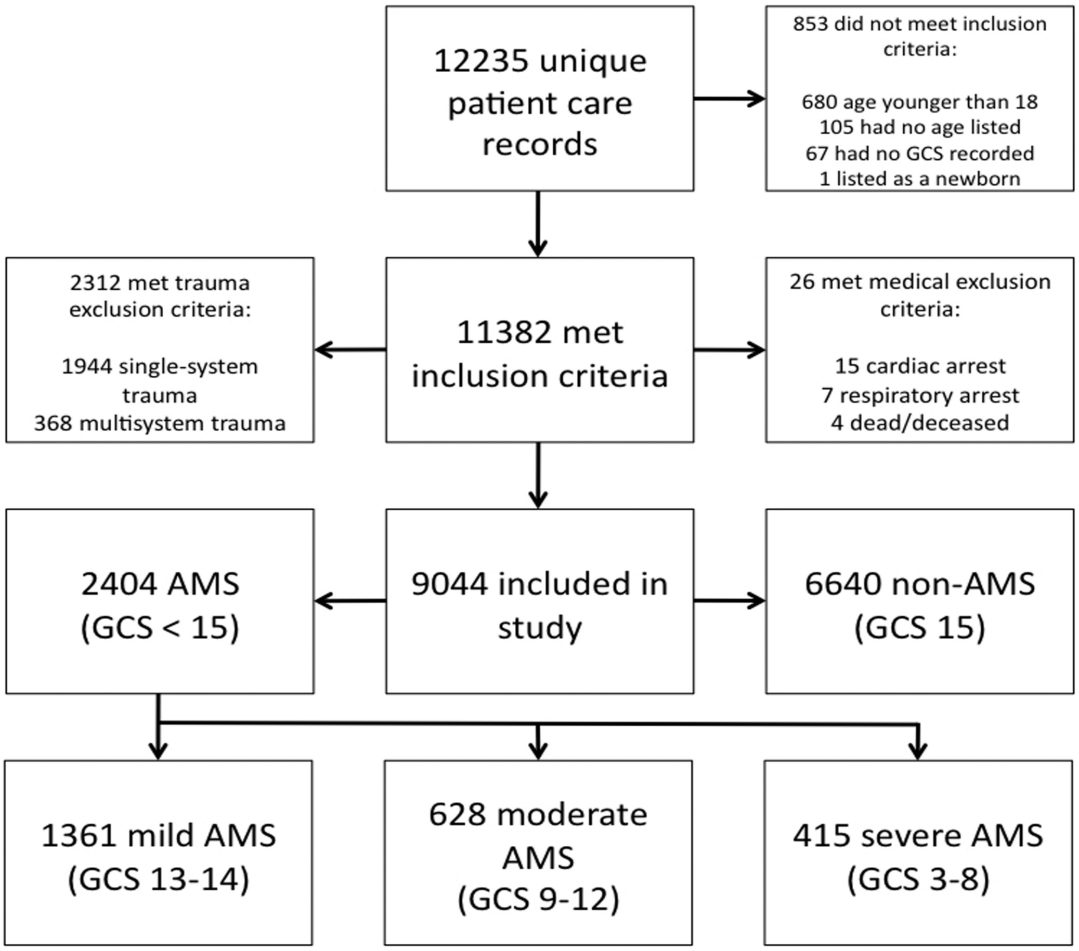

Figure. Cohort flow diagram.
GCS, Glasgow Coma Scale; *AMS,* altered mental status

responses. The three values are considered separately and summed. The lowest possible GCS is three (deep coma or death), while the highest is 15 (fully alert and oriented). With good interobserver reliability and ease of use, we linked the admission GCS to prognosis prediction for a number of conditions, including risk for aspiration pneumonia, traumatic brain injury, subarachnoid hemorrhage and bacterial meningitis.[6-9] Furthermore, prior studies validate paramedic GCS use, demonstrating good inter- and intraobserver reliability, and statistically significant reliability between paramedics and emergency physicians.[10]

A checkbox format that included 22 medical conditions documents past medical history (PMH). A checked box indicated a known condition in the PMH, while an unchecked box indicated either "none" or "unknown" (no distinction was made). Of the conditions listed, six are known causes of ALOC: stroke/transient ischemic attack (TIA), diabetes (due to episodes of hypoglycemia), alcohol use, psychiatric illness, illicit drug use and seizures.[11,12] We excluded trauma patients with ALOC from our analysis. We compared whether the abnormal and normal GCS groups differed statistically in the proportion with a known ALOC etiology or no PMH. The statistical package used for data analysis was SAS 9.1.3, Cary, NC.

RESULTS

We examined 12,235 unique PCRs, of which 9,044 (73.9%) met inclusion criteria. We excluded only 67 adult patients (0.59%) for having no documented GCS. The normal GCS group contained 6,640 (73.4%) patients and 2,404 (26.6%) abnormals (Figure). The mean age of the abnormal GCS patients was 65.6 years, compared with 61.4 in the normal GCS (Table 1). We associated each ten-year increase in age with an increased likelihood of abnormal GCS (odds ration [OR]=1.1, 95% confidence interval [CI]=1.08-1.12). Abnormal GCS patients were more likely to be male (50.5%) than normal GCS patients (43.7%; OR=1.31, CI=1.19-1.44).

In the abnormal GCS group, 415 (17.3%) patients were severe (GCS 8 or less), 628 (26.1%) moderate (GCS 9-12) and 1,361 (56.6%) mild. The mean age in each group was 72.0, 75.0, and 68.0, respectively. There was no statistically significant association between age and severity of abnormal GCS. Male sex was more likely to be associated

Table 2a. Percentage of patients by group receiving intervention

	GCS 15	GCS 3-14
IV access (%)*	61.7	75.7
Medications administered (%)	32.0	32.3
Activated charcoal	0.69	0.42
Albuterol	6.3	6.8
Glucose, oral*	1.1	2.0
Dextrose, IV*	0.8	9.0
Glucagon*	0.03	1.2
Midazolam*	0.17	2.4
Naloxone*	0.18	5.3
Nitroglycerin*	9.5	2.2

**Indicates statistically significant result, (p<0.05)*
GCS, Glasgow Coma Scale; *IV,* intravenous

with a mild abnormal GCS (OR=1.52, CI=1.35-1.71). There was no statistically significant association between male sex and either moderate or severe abnormal GCS. There were no significant differences in race between the two groups (Table 1).

The most common primary paramedic diagnostic impressions in the abnormal GCS group were ALOC (32.3%), postictal state (10.0%), "other" (7.2%), hypoglycemia (6.3%) and syncope/near-syncope (5.9%). In the normal GCS group, they were "other" (14.7%), weakness (13.9%), syncope/near-syncope (9.0%), abdominal pain (8.6%) and non-traumatic body pain (7.3%).

Patients in the abnormal GCS group were more likely to have an IV placed (OR=1.94, CI=1.74-2.2). Although these patients were no more likely to receive medications in the field (OR=1.01, CI=0.92-1.12), patients with a mildly abnormal GCS were less likely to receive medications, while those with a moderately or severely abnormal GCS were more likely (Table 2a). In the abnormal GCS group, patients were no more likely to receive activated charcoal than the normal GCS group, and only twice as likely to receive oral glucose compared with the normal GCS group. The rates of administration for parenteral medications in the abnormal GCS group, compared with the normal GCS group, were dramatically different, with abnormal GCS patients significantly more likely to receive glucagon, midazolam or naloxone (Tables 2a and 2b). The transport rates were similar in both groups (92.5% in the abnormal vs. 90.0% in the normal GCS group).

A known PMH was documented in 43.1% of patients (45.9% of abnormal GCS and 42.1% of normal GCS). The remainder of patients had either no known, or an unknown PMH, though this was not differentiated in the paramedic documentation. The most common PMH for abnormal GCS patients were hypertension (25.2%), diabetes (14.4%), seizures (6.9%), stroke/TIA (6.6%) and non-specific cardiac (5.8%). For normal GCS patients, they were hypertension (25.2%), diabetes (11.1%), non-specific cardiac (5.5%), asthma (5.1%) and congestive heart failure (4.9%).

In comparing known causes of ALOC in the PMH, we found that patients with a PMH of stroke or TIA, diabetes, alcohol use, substance abuse and seizures all were significantly more likely to present in the abnormal GCS group. We found no association, however, between a past psychiatric history and an abnormal GCS (Table 3a). All of the ALOC-associated conditions were positively associated with the male sex and older age. Controlling for age and sex, however, did not significantly affect the results. Additionally, having no known PMH, or an unknown PMH, was negatively associated with an abnormal GCS (OR 0.85, CI=0.78-0.94).

We subdivided the abnormal GCS group into mild (GCS 13-14), moderate (GCS 9-12) and severe (GCS 3-8) categories (Table 3b). Patients with alcohol use and seizures in their PMH trended toward higher GCS and patients with diabetes in their PMH trended toward lower GCS. Patients with stroke/TIA or substance use in their PMH had a higher likelihood of presenting in the moderately altered group, with neither group showing a trend toward high or low GCS. Adjusting for age and sex had minimal effects on the results (Table 3c). The only statistically significant change was a new association between mild abnormal GCS and psychiatric illness.

DISCUSSION

Patients with an abnormal GCS tended to be older and male and increasing age correlated with an increasing likelihood of an abnormal GCS. The higher prevalence of certain ALOC-associated conditions among males (alcoholism and illicit drug use) revealed a potential confounding source, and the association of older age with stroke and diabetes likely demonstrates effect modification.[13,14] In our study, all ALOC-associated conditions except psychiatric illness were positively associated with male sex and older age-However,

Table 2b. Odds ratios and 95% confidence intervals for paramedic interventions by GCS group

Intervention	GCS 13-14	GCS 9-12	GCS 3-8
IV access	1.57 (1.38-1.78)	2.25 (1.85-2.73)	3.60 (2.73-4.75)
Medications	0.61 (0.53-0.70)	1.27 (1.07-1.50)	2.86 (2.34-3.49)

All results were statistically significant (p<0.05)
GCS, Glasgow Coma Scale; *IV,* intravenous

Table 3a. Odds ratios and 95% confidence intervals for known altered mental status etiologies in past medical history

	Unadjusted odds ratio	Adjusted for age and sex
Alcohol*	2.3 (1.75-3.00)	2.60 (1.95-3.40)
Diabetes*	1.34 (1.17-1.54)	1.25 (1.09-1.44)
Psychiatric	1.00 (0.78-1.29)	1.17 (0.91-1.52)
Seizures*	3.00 (2.40-3.80)	2.50 (2.80-4.40)
Stroke/TIA*	2.00 (1.64-2.50)	1.79 (1.45-2.20)
Substance use*	1.60 (1.09-2.30)	1.86 (1.26-2.70)

**Indicates statistically significant result, (p<0.05)*
TIA, transient ischemic attack

the association between known ALOC etiologies in the PMH and older age tended to become statistically significant only in the oldest quartile of our study group (age 82-104). We found no statistically significant associations between race and abnormal GCS.

An association exists between a known ALOC etiology in PMH and the presentation of an abnormal GCS in the field. Although this finding is not surprising, the number of patients without an unknown PMH or with no known PMH was unexpected. With the advent of binary PMH checkboxes and computerized PCRs, practitioners often miss the distinction between not having a PMH and having an unknown PMH. This is an area of quality control that could be addressed in the future.

In our study population, we identified diabetic patients as among the most common, as well as the most likely to present with a severely abnormal GCS. Although a blood glucose check is standard practice in the patient with an abnormal GCS, given the short and long-term sequelae associated with hypoglycemia, such as increased risk of myocardial infarction, ventricular rhythm disorders, and stroke, perhaps blood glucose monitoring should be given a higher priority in paramedic protocols when treating the severely altered non-trauma patient.[15]

Another unexpected finding was that while only 38 patients (1.6% of the abnormal GCS group) presented with a persistent seizures or status epilepticus, 2.4% of them received midazolam. San Mateo County paramedic protocols indicate the use of midazolam only for persistent seizures and pre-cardioversion sedation (a relatively rare event). These results likely indicate a significant amount of "off-label" use (e.g., for sedation in agitated patients with base hospital approval). This is in an area where the protocols could be revised to reflect actual field use.

As a group, abnormal GCS patients required more

Table 3b. Unadjusted odds ratios and 95% confidence intervals for known altered level of consciousness etiologies in past medical history by Glasgow Coma Scale (GCS) category.

	GCS 13-14	GCS 9-12	GCS 3-8
Alcohol	2.95 (2.18-4.00)*	2.01 (1.26-3.19)*	0.67 (0.27-1.66)
Diabetes	1.01 (0.84-1.21)	1.68 (1.35-2.10)*	2.04 (1.58-2.62)*
Psychiatric	1.24 (0.92-1.65)	0.67 (0.39-1.13)	0.74 (0.40-1.37)
Seizure	3.44 (2.67-4.43)*	2.48 (1.71-3.59)*	2.50 (1.61-3.89)*
Stroke/TIA	1.84 (1.42-2.38)*	2.81 (2.07-3.81)*	1.45 (0.91-2.32)
Substance use	1.82 (1.17-2.85)*	1.75 (0.95-3.25)	0.66 (0.21-2.09)

**Indicates statistically significant result, (p<0.05)*
TIA, transient ischemic attack

Table 3c. Age and sex adjusted odds ratios and 95% confidence intervals for known altered level of consciousness etiologies in past medical history by Glasgow Coma Scale (GCS) category.

	GCS 13-14	GCS 9-12	GCS 3-8
Alcohol	3.11 (2.28-4.25)*	2.55 (1.59-4.08)*	0.80 (0.32-1.97)
Diabetes	0.95 (0.79-1.14)	1.53 (1.22-1.91)*	1.91 (1.48-2.45)*
Psychiatric	1.40 (1.04-1.89)*	0.84 (0.49-1.43)	0.88 (0.47-1.63)
Seizure	3.84 (2.96-4.97)*	3.10 (2.12-4.52)*	2.95 (1.89-4.61)*
Stroke/TIA	1.67 (1.28-2.18)*	2.40 (1.76-3.27)*	1.25 (0.78-2.01)
Substance use	1.99 (1.27-3.13)*	2.30 (1.23-4.28)*	0.79 (0.25-2.53)

**Indicates statistically significant result, (p<0.05)*
TIA, transient ischemic attack

paramedic medical interventions than those with a normal GCS. Patients with an abnormal GCS were more likely to have IV access established by paramedics. Although the rates of medication administration were no different overall between the normal and abnormal GCS groups, mild GCS was associated with lower rates of medication administration than normal GCS, while moderately and severely abnormal GCS were each associated with increasing rates of medication administration. Although existing San Mateo County EMS protocols direct paramedic interventions, routine IV access is not required for ALOC patients.[16] Previous studies demonstrated that most paramedics initiated IV lines are not used to provide treatment, such as fluids or medications in the field.[17] This seemed to hold true for patients with a mildly abnormal GCS, who were more likely to receive an IV and less likely to receive medications. Patients who were categorized in the moderately and severely abnormal GCS groups, however, had smaller relative odds ratios of receiving an IV compared with receiving medication, indicating that in these groups, IVs were more likely to be medically necessary rather than precautionary.

San Mateo County paramedics carry 18 different medications, but paramedics only used a third of these (activated charcoal, dextrose, glucagon, glucose paste, midazolam and naloxone) in the treatment of known ALOC etiologies. Of the 58 different paramedic diagnostic impressions listed in the database, only a dozen are commonly associated with ALOC (excluding cardiopulmonary arrest). Of these, only hypoglycemia and overdose routinely require medication administration while seizures require medication administration only if the patient is actively seizing. Hypoglycemia, however, was relatively common in the abnormal GCS group (6.3%) while medical conditions in the normal GCS group that routinely required medication administration were relatively rare.

We found five of the six conditions in the PMH known to be associated with ALOC to have an association with an abnormal GCS in our study. Seizures and alcohol use indicate the strongest associations with an abnormal GCS, while associations between an abnormal GCS and stroke/TIA, diabetes and substance abuse were weaker. We did not find the expected association between a history of psychiatric illness and an abnormal GCS. This may be partly attributable to the difficulty of obtaining an accurate psychiatric history from an altered patient. There was, however, a statistically significant negative association between having no listed PMH ("null" category) and an abnormal GCS (OR 0.85, CI=0.78-0.94, p=0.0011). Given the higher prevalence of psychiatric illness among alcoholics and illicit drug users and the difficulty of obtaining a medical history from an altered patient, it is possible that paramedics presumed only a history of alcohol or substance abuse, but not psychiatric illness, in the intoxicated patient.[18,19] Thus, psychiatric illness may have been underreported in this group.

LIMITATIONS

A major limitation of this study is that it was conducted in a single county in California. These results would not necessarily generalize to populations in a more rural or more urban setting than San Mateo County. Furthermore, as paramedic interventions are protocol-directed, the rates and types of interventions may differ in counties with more or less aggressive protocols and medical direction.

One limitation of this study is the exclusion of trauma patients from our analysis. This was done for several reasons, including the generally non-episodic nature of trauma, a lack of causality between prior and subsequent trauma, and the often multifactoral etiology of ALOC associated with trauma. Other studies validated the use of the GCS for measuring ALOC in trauma patients.[20]

Another major limitation of this study is that the "null" category in the PMH could mean either that the patient had none or that the patient's PMH was unknown. Given the often episodic nature of ALOC, a negative association between having no PMH and ALOC seems reasonable. The degree of that association, however, was likely masked by the number of patients in the "null" category who actually had a PMH causally contributing to their abnormal GCS was not known, and could not be obtained by the paramedics.

The GCS does not distinguish between the various causes of ALOC, and similar GCS does not indicate a similar etiology. The GCS is particularly poor at measuring agitated and sedated states, which have important diagnostic and prognostic value in altered patients.[21]

The exclusive use of prehospital data limited the scope of our study. Further studies linking prehospital data with ED data would be needed to answer more sophisticated questions on the diagnostic and predictive value of the GCS in non-trauma patients.

CONCLUSION

Patients with an abnormal GCS differ from patients with a normal GCS in terms of age, sex, paramedic field impression, past medical history and prehospital treatment. Patients with an abnormal GCS tend to be older and male, and excluding a history of psychiatric illness, are more likely to have a known ALOC etiology in their PMH. Although the GCS is useful in assessing the level of impairment in arousal, it is of limited usefulness in determining the etiology of ALOC. For these reasons, future prospective studies should focus on subgroups within this diverse patient population, particularly diabetics and the elderly, to help elucidate optimal prehospital management.

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Mass Casualty Incident Response and Aeromedical Evacuation in Antarctica

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Antarctica is one of the most remote regions on Earth. Mass casualty incident (MCI) responses in Antarctica are prone to complications from multiple environmental and operational challenges. This review of the current status of MCI risks and response strategies for Antarctica focuses on aeromedical evacuation, a critical component of many possible MCI scenarios. Extreme cold and weather, a lack of medical resources and a multitude of disparate international bases all exert unique demands on MCI response planning. Increasing cruise ship traffic is also escalating the risk of MCI occurrence. To be successful, MCI response must be well coordinated and undertaken by trained rescuers, especially in the setting of Antarctica. Helicopter rescue or aeromedical evacuation of victims to off-continent facilities may be necessary. Currently, military forces have the greatest capacity for mass air evacuation. Specific risks that are likely to occur include structure collapses, vehicle incapacitations, vehicle crashes and fires. All of these events pose concomitant risks of hypothermia among both victims and rescuers. Antarctica's unique environment requires flexible yet robust MCI response planning among the many entities in operation on the continent. [West J Emerg Med. 2011;12(1):37-42.]

BACKGROUND

Antarctica, covered almost entirely by massive sheets of ice, is one of the most remote and challenging environments on Earth.[1] Its importance in scientific research and exploration has led many nations to develop interests in the region. The Antarctic Treaty, in effect since 1961, has 47 signatory nations that assert their dedication to maintain Antarctica as a continent devoted to peaceful and scientific pursuits. Any military operations are restricted to peaceful support missions.[2]

Approximately 30 nations operate more than 80 research stations in the region, mostly near the coast. Nearly half of the stations operate only during the summer months. On the central plateau, only the United States' (U.S.) South Pole Station operates year-round through the dark six months of winter.[3]

Tourism represents a relatively new development in the Antarctic environment. Tourism is rising significantly around the region on a yearly basis, and the risk of tour boat disasters is therefore increasing. Most ships carry medical personnel on board but have limited resources for dealing with a mass casualty situation.

OBJECTIVE

This paper reviews the current status of Antarctic mass casualty incident (MCI) risks and response strategies from the perspective of aeromedical evacuation. Due to its remoteness, evacuation of victims by air, both within and from the Antarctic region, is a critical component of a wide variety of response scenarios following a future MCI.

CHALLENGES TO MCI RESPONSE IN ANTARCTICA

Environment

An MCI is a disaster that overwhelms local resources, which in Antarctica are already scarce. The Antarctic environment includes sub-zero temperatures, seasons of continuous darkness, and extraordinary storms, all of which require unique measures of preparedness.[5]

Although the potential for a disaster is present year-round, a large-scale MCI would be most likely to occur during the summer months of November through February because there are considerably more people in the region - approximately 5,000 on the continent and tens of thousands of tourists in the surrounding waters.[6] The largest Antarctic base, the U.S.'s

McMurdo Station, holds approximately 1,400 people during the summer.

During the winter months, Antarctica experiences continuous darkness. Temperatures routinely sink below -50°C, as opposed to slightly below freezing in the summer. In winter there are about 1,000 inhabitants on the continent and essentially no tourists.[7]

Another challenge imposed by the Antarctic environment is that magnetic navigation is complicated by its proximity to the South Magnetic Pole, which is currently off the Antarctic coast in the Eastern Hemisphere. The Global Positioning System satellite network has greatly improved accurate navigation.

Distance

Distances between Antarctica and civilization are vast, which is perhaps the most formidable of all the challenges listed. Travel from McMurdo Station to the nearest city, 2,400 miles away in New Zealand, takes five hours by plane. The bases of the various nations are scattered, rudimentary and separated by thousands of miles of hostile expanse. This degree of isolation is unmatched by any other human settlement and causes inevitable delays and difficulties in mounting an MCI response.

Increasing Cruise Ship Tourism

Most Antarctic cruises embark from Ushuaia, Argentina, the world's southernmost city. Argentina and Chile have few facilities to assist distressed ships in the Antarctic area, although their Joint Antarctic Naval Patrol maintains a dedicated search and rescue presence every summer.[8] Ships in Antarctic waters run aground, become trapped in pack ice, sink, catch fire, or break down on a yearly basis in the Southern Ocean. The nearest rescue vessels may be a day or more away. Although casualties have been rare to this point, thanks to well-provisioned lifeboats and emergency plans, a foundering ship could potentially create an enormous disaster in Antarctic waters.

Approximately 40,000 people visited Antarctica during the 2008-2009 season, representing a 400% increase relative to 14 years prior.[9] Antarctic cruise ships are also getting larger; some carry up to 4,000 passengers. It would be difficult to rescue victims from a ship of that size, as most rescuing ships are far smaller and have limited capacity. The International Association of Antarctic Tour Operators (IAATO) has recently barred Antarctic landings for ships carrying more than 500 passengers; however, these guidelines are not internationally binding for non-IAATO members.

Prevention

The extreme difficulty in MCI response in Antarctica requires conscientious prevention measures. Strategies for preventing MCIs include extensive safety and training protocols and specialized equipment to withstand the climate. Additionally, in the Antarctic Ocean, melting ice has opened new, uncharted coastal waters, and the Antarctic Treaty Consultative Meeting (ATCM) has identified improved Antarctic hydrography as a critical measure to prevent an MCI at sea.[10]

MASS CASUALTY INCIDENT RESPONSE

MCI Success

A successful MCI operation relies on clear, well-practiced protocols, good communication networks, and access to sufficient resources.[11,12] When resources are overwhelmed, those unlikely to survive without immediate intensive care should not be resuscitated or transported, especially in an environment with such sparse resources and dangers from exposure as Antarctica.[13,14] The capacity of the clinic or staging area for victims may be quickly exceeded, necessitating consideration of all available warm, lighted areas as potential treatment centers. Trauma victims not killed immediately during an MCI often succumb to a combination of hypovolemia, acidosis and coagulopathy within the first 24 hours. An additional concern in Antarctica is hypothermia, and patients in open areas are at particular risk. Resuscitation of critically injured patients awaiting evacuation must focus on rewarming and adequate early resuscitation to the extent feasible.

Coordination

Antarctica and the surrounding ocean are divided into five Search and Rescue Regions, each under the jurisdiction of a Rescue Coordination Center (RCC), based in New Zealand, Australia, South Africa, Argentina and Chile. Emergency radio beacons, coordinated by satellite using the Global Positioning System that can relay a signal to the nearest RCC, are mandatory on most sea and air vessels. Use of beacons could minimize the search phase of a search and rescue operation for a missing vehicle, thereby improving MCI response.[15] For a ship or aircraft in distress, the RCC is tasked with locating vessels capable of responding and has authority to impel them by UN convention to assist with rescue efforts.[16]

Unlike maritime and aeronautical response to distress signals, there are no official agreements for cooperation in Antarctic land-based MCIs,[17] although any nation with capacity to assist in an emergency can be reasonably expected to do so. Responsibility for land-based search and rescue is traditionally considered to be primarily that of the national Antarctic Program involved. The Antarctic Programs of other nations and the nearest of the five RCCs would be expected to provide assistance as required.[15]

The Antarctica Search and Rescue Workshop was created in 2008 to coordinate planning and response to an Antarctic MCI; they had their second meeting in 2009.[15, 17] One goal of the workshop is to develop international agreements for land-based MCIs that have not yet been solidified. The yearly ATCM provides a forum for codifying

such agreements.[9,10] Although objective preparedness for an unknown future event is difficult to measure, the workshops have provided for clearer understanding between bases of relative rescue capabilities, as well as analysis of multiple actual and hypothetical disaster scenarios from a collaborative perspective of all stakeholders.[17]

AEROMEDICAL EVACUATION

Short-Range Aeromedical Evacuation

An MCI occurring far from an Antarctic base may require rotary aircraft to transport patients to the nearest base for initial stabilization. Most bases have one or two helicopters in operation; McMurdo, the largest base, has four.[18] Most helicopters in Antarctica are AS350 Squirrels and Bell 212s, which are both versatile and suited to rescue operations. Maritime vessels occasionally carry a helicopter as well.

Long-Range Aeromedical Evacuation

In MCIs most lives are initially saved by survivors and bystanders after an incident, rather than by medical crews. Air evacuation should therefore focus on decreasing mortality among the early survivors who are at risk of delayed death.[19] This group is most likely to die from complications following trauma, such as internal hemorrhage, sepsis or multi-organ failure. Transferring these patients to advanced medical facilities can greatly increase their chance of survival.[19] The most advanced medical facility on Antarctica is McMurdo General Hospital (Figure), which is essentially a basic clinic with little critical care capacity and no surgical capability.[20]

Remote Aeromedical Response

Aeromedical responses to remote locations require more strategy and planning than responses to populated areas because there are fewer resources to rely on upon arrival.[21] Difficulties in remote air evacuations potentially arise with communications, division of responsibility, unfamiliar terrain, equipment problems, personnel problems, crew exhaustion, inclement weather and the difficult clinical decisions one is forced to make when faced with exiguous resources.[19,22] In Antarctica, many types of aircraft cannot easily land on the continent's rough-hewn ice runways, so long-range Antarctic operations are accomplished by specialized military aircraft such as American C-130s and C-17s that have been fitted with ski-type landing gear.

Figure. McMurdo General Hospital. Photo courtesy of Ken Iserson, MD.

Military Response Capacity

Militaries are under no statutory obligation to evacuate civilians. Nevertheless, most of the aeromedical evacuations from Antarctica in the past have been accomplished by military teams, and it can be assumed that military branches would deploy to play a crucial role in aeromedical response for an Antarctic MCI. International cooperation is critical for success.[23] The U.S. employs a system of Critical Care Air Transport Teams (CCATT), each consisting of a critical care physician, a critical care nurse and a respiratory therapist.[24,25] Although surgical intervention is not feasible en route, the CCATT team operates as a portable intensive care team within a cargo aircraft. The most commonly used aircraft for the team is the C-130 Hercules, which has the capability to land on unimproved airfields and carry up to 74 litter patients. The C-130 is routinely flown to Antarctica with affixed skis. Another aircraft commonly used is the C-17 Globemaster. This aircraft is better suited to patient transfer than the C-130. Although it can only carry 36 litter patients, it flies higher and faster than the C-130 and offers better lighting and electrical power. It also improves patient transport with a warm environmental system and superior access to patients from both sides.

Airdrops

Aeromedical resources may also be used to deliver critical supplies via airdrop if landing is not possible. In the winter, cold and darkness preclude landing. In the winter of 1999 airdrops over South Pole station were used to deliver chemotherapy and biopsy equipment to the station's physician, who diagnosed herself with breast cancer during her winter over.[26] In 2007, an airdrop delivered engine parts to a disabled trawler in Antarctic waters, demonstrating this as a possible strategy in a maritime MCI as well.

Innovations in Aeromedical Response

Several advances in technology could hold promise for future Antarctic MCI responses. One such innovation is a mobile trauma center in the space of a 53-foot trailer that can fit in the cargo bay of a C-130J-30 (a lengthened C-130 cargo plane). This trailer is equipped with six critical care beds and seven noncritical beds.[27] An attached tent system can add 100 exterior beds, although low temperatures in Antarctica could limit the applicability of this feature. The trailer could be deployed as a stand-alone unit to respond to an MCI far from

base or as a critical care overflow area at the base. Bringing such medical resources to Antarctica could become necessary in a massive MCI where timely aeromedical evacuation is not possible. Adding heating units, a capable tow vehicle, and a specialized suspension could make this type of resource invaluable.

For rapid aeromedical evacuation, the Swedish National Air Medevac (SNAM) can convert any standard passenger airliner with no specialized features into an aeromedical evacuation aircraft within six hours by replacing seats with efficiently-designed intensive care beds and litter racks. After the 2004 Indian Ocean tsunami, SNAM outfitted three airliners with 92 beds that brought hospitalized patients back to Sweden five days post-disaster.[28] A similar strategy could be used in Antarctica if military evacuation capacity is exceeded or unavailable; however, attachment of skis for ice runway landings may be a complicating factor.

MCI SCENARIOS IN ANTARCTICA

Mass Impact or Structure Collapse

Mass trauma due to a large impact (such as from falling objects, snapping cables or malfunctioning machinery) or building collapse has yet to occur on the Antarctic continent. Although earthquakes are relatively uncommon in Antarctica, they do occur. In both 1998 and 2004, for example, very large earthquakes of magnitude 8.1 on the Richter scale struck off the coast.[29,30] If structures collapse, extrication and sheltering efforts may be largely unassisted for a long period of time, exposing victims to dangerously cold temperatures in addition to their injuries.

Hypothermia threatens to strike rescuers as well as victims.[31] The more practiced the local population is with outdoor rescue operation drills, the greater the chance of successful rescue when an MCI strikes, and the lower the chance of responders becoming victims themselves.

Vehicle Incapacitation

Ships, aircraft, and land-based vehicles are in use all over the Antarctic region. All are at risk of becoming remotely stranded or incapacitated. One such example includes the first cruise ship designed for Antarctica, the *MS Explorer*. The *Explorer* sank after striking ice in 2007.[4] Three vessels were within 40 miles of the vessel and were activated by the Argentine RCC to respond immediately. More than 150 passengers and crew were rescued from the stricken ship.[32] They were then transported to the nearest Antarctic base, from which they were flown to Punta Arenas in southern Chile. The evacuation took place in favorable weather and typical air temperatures of about -5°C. The only medical consequences were four cases of moderate hypothermia.

Much of the Antarctic Ocean remains uncharted, and coastal water is increasing as polar ice melts more than it refreezes each year. Ships face a multitude of hidden hazards including land, shoals, and icebergs. Increasing vessel traffic has increased the risk of an MCI. Sudden calving of ice shelves can also pose a potential hazard to ships.

Helicopters may be very useful to locate and rescue victims. Although a sinking ship may be the most likely disaster scenario in Antarctica, long-range aeromedical evacuation in this situation has a limited role. In most situations, a ship in distress incurs few traumatic casualties and allows adequate time for lifeboat deployment. The largest risk is hypothermia, which is unlikely to benefit from air transport to a hospital off-continent.

Vehicle Crash

Air New Zealand flight 901 crashed into an Antarctic mountainside in November 1979 while conducting a routine sightseeing flight.[33] Weather conditions created a white-cloud backdrop behind the snow-covered Mount Erebus, creating a "sector whiteout." The aircraft's ground proximity warning sounded and the crew responded appropriately with upward pitch and full power, but it was too late. Six seconds later, a full-speed impact occurred, killing all aboard. Search aircraft found the wreckage 12 hours after the crash. Had there been evidence of any survivors, helicopter crews would likely have been deployed from New Zealand's Scott Base and the US's McMurdo Station, both about 40 miles from the crash site.

Another plane crash in 2007 resulted in only minor injuries.[34] In this case, the crash survivors were fortunate to have good weather, intact survival gear, functioning communications equipment and minimal injuries.

The potential for a vehicle crash is a real threat on the Antarctic continent. Land-based vehicles contend with uncertain terrain and poor visibility conditions. Aircraft face problems such as difficulty judging height above blinding white terrain, a runway system largely made up of unfinished, rutted ice fields, scant aeronautical resources, daunting weather conditions and extreme remoteness. Vehicle crashes in particular have the potential to cause MCIs very distant from any clinical resources. Helicopter rescue to the nearest base is the most likely response in this scenario. Alternatively, setting up a field stabilization center at a remote scene may be necessary for a large-scale MCI response; however, freezing weather conditions could render this strategy untenable.[35]

Explosion or Fire

In Antarctica, the fuel caches and power generators that support the inhabitants are at risk of explosion. Structure fires and vehicle explosions also pose a risk of MCI. Water quickly freezes, requiring specialized chemicals to fight fires. Antarctica is a desert with very dry air that allows fire to spread quickly despite the cold. Occasional fires have occurred in Antarctica in the past. A large fire at the UK's Rothera Station in 2001 destroyed the laboratory. Luckily, there were no casualties. A Japanese whaling ship caught fire in 2007 off the coast, killing one person.[36] Also, in 2008 a fire at Russia's Progress Station killed one and injured two

people. Beyond the danger from injuries and burns, loss of an entire base to fire would be a devastating event because of the immediate threat of lethal hypothermia. McMurdo Fire Department and its counterparts at other bases are highly focused on prevention measures.

CONCLUSION

A wide variety of potential MCIs threaten Antarctica, and advanced planning is crucial to ensure the health and safety of the continent's population.[37] Rapid communication of the situation, conversion of local resources into triage centers,[35] efficient transportation of victims to base clinics,[38] consideration of supply and trauma team delivery to the MCI site, and air evacuation to off-continent medical facilities are all cornerstones for an effective Antarctic MCI response.[39,40] Coordination between National Antarctic Programs and the five southernmost RCCs to these ends will improve chances for victim survival in the aftermath of a future Antarctic disaster.

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Analgesia for Older Adults with Abdominal or Back Pain in Emergency Department

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Objective: To determine the association between age and analgesia for emergency department (ED) patients with abdominal or back pain.

Methods: Using a fully electronic medical record, we performed a retrospective cohort study of adults presenting with abdominal or back pain to two urban EDs. To assess differences in analgesia administration and time to analgesia between age groups, we used chi-square and Kruskal-Wallis test respectively. To adjust for potential confounders, we used a generalized linear model with log link and Gaussian error.

Results: Of 24,752 subjects (mean age 42 years, 65% female, 69% black, mean triage pain score 7.5), the majority (76%) had abdominal pain and 61% received analgesia. The ≥80 years group (n=722; 3%), compared to the 65-79 years group (n=2,080; 8%) and to the <65 years group (n=21,950; 89%), was more often female (71 vs. 61 vs. 65%), black (72 vs. 65 vs. 69%), and had a lower mean pain score (6.6 vs. 7.1 vs. 7.6). Both older groups were less likely to receive any analgesia (48 vs. 59 vs. 62%, p<0.0001) and the oldest group less likely to receive opiates (35 vs. 47 vs. 44%, p<0.0001). Of those who received analgesia, both older groups waited longer for their medication (123 vs. 113 vs. 94 minutes; p<0.0001). After controlling for potential confounders, patients ≥80 years were 17% less likely than the <65 years group to receive analgesia (95% CI 14-20%).

Conclusion: Older adults who present to the ED for abdominal or back pain are less likely to receive analgesia and wait significantly longer for pain medication compared to younger adults. [West J Emerg Med. 2011;12(1);43-50]

INTRODUCTION

Acute pain is one of the most common reasons for presenting to an emergency department (ED).[1] Oligoanalgesia, or the undertreatment of pain, has been studied in the emergency setting and is well documented in the emergency medicine (EM) literature.[2-4] Disparities in pain management have been attributed to a variety of patient factors, including gender, race, insurance status and extremes of age.[5-14] Pain management has been identified as a specific issue for quality-of-care improvement in older adults.[15,16] In emergency care, quality indicators that include effective analgesia have been developed for pain management in older adults as they are less likely to receive analgesia than younger adults with similar conditions.[17] A study of 231 ED patients with isolated long-bone fractures showed that older adults were less likely to receive analgesia when compared with younger adults with similar injuries. Additionally, those older adults who received analgesia waited longer for their medication and were less likely to receive an opioid analgesic.[12]

Oligoanalgesia in older adults may be related to both provider and systems factors. In a survey of emergency physicians, almost half of providers reported discomfort with

their degree of training in geriatric EM and in the practice of prescribing analgesia to older adults.[18] Recently, an expert consensus process has identified geriatric pain management as a minimal core competency for EM residents.[19] Factors affecting the ED as a whole may also contribute to oligoanalgesia; Hwang et al.[20] found that increased crowding in the ED led to underassessment and undertreatment of pain, delays in analgesia and treatment with inappropriate pain medications in older ED patients with hip fractures.

In 2006 adults age 65 and older accounted for over 17.3 million ED visits, or approximately 15% of all visits.[1] However, based on predicted demographic changes, older patients will increase to as much as one-third of all ED visits in the next 30 years.[21, 22] Older adults use emergency services at much higher rates and have longer stays in the ED than younger adults.[23] Older adults are also a vulnerable population in the emergency care setting,[24] with data demonstrating inferior quality of care leading to poor clinical outcomes.[25-29] Specifically, oligoanalgesia in older patients may also lead to negative consequences such as delirium, depression, and increased hospital length of stay.[30-32]

Abdominal pain and back pain are two of the most common specific reasons for seeking emergency care, with abdominal pain as the leading reason to visit the ED accounting for eight million visits annually.[1] While historically the early use of analgesia in ED patients with abdominal pain was thought to mask signs of peritonitis and potentially delay care, multiple well-designed randomized controlled trials have conclusively shown that this practice does not lead to adverse outcomes.[33-41] The Joint Commission's 2001 pain management standards state that every patient has a right to have his or her pain assessed and treated.[42] Therefore, the timely use of analgesia in patients who have been assessed appropriately and request symptom control should be a standard of care in EM practice. Analgesics, specifically opiate analgesics, have been shown to be safe for older adults when understood and used correctly.[43-45] To our knowledge, there have been no large studies examining the effect of age on the administration of analgesia for acute pain in an emergency setting. We sought to study the affect of age on a large cohort of more than 24,000 patients presenting to the ED with abdominal or back pain. We hypothesized that older adults would be less likely to receive analgesia for the treatment of these complaints.

METHODS

Study Design and Setting

We performed a retrospective cohort study of adult ED patients who presented with a chief complaint of abdominal pain or back pain. The study was performed at the Hospital of the University of Pennsylvania (HUP) and Presbyterian Medical Center (PMC), two urban EDs with 57,000 and 35,000 annual visits respectively. The Institutional Committee on Research Involving Human Subjects at the University of Pennsylvania approved the study.

Selection of Participants

We identified patients using an electronic medical record search of EMTRAC (University of Pennsylvania, Philadelphia, PA), a comprehensive computerized charting system. We included all adult ED patients, 18 and older, who presented between July 2003 and February 2007 to triage with a chief complaint of abdominal pain or back pain. Specific chief complaint variants of abdominal pain (e.g., gastroesophageal reflux disease, urinary tract infection, etc) or back pain complaints (e.g., sciatica, muscle spasm, etc) were not included. Patients were excluded who left the ED without being seen, who were pregnant, or who had no documented pain score or a pain score of "0." Patients were categorized into one of three groups: adults ≥80 years of age, adults between the ages of 65 and 79, and adults <65 years of age.

Methods of Measurement

We created a database from EMTRAC, a fully electronic emergency medical record, where each entry, order, or activity is automatically time-stamped for pre–specified ED events (e.g., when a patient is first seen by a nurse in triage; is placed in an ED room; orders are entered by a physician; completion of medication orders by a nurse). The database included patient demographics (age, sex, race), triage classification (4-point scale [1-4] from emergent [1] to nonemergent [4]), triage pain score, pain medication type and time of administration and final ED disposition. Triage pain scores were documented using an 11-point scale (0-10), from "no pain" (0) to "worst pain of my life" (10). For this study we excluded patients with a pain score of "0." Reassessments for pain were not available for this analysis. We obtained the type and time of the first analgesic administration directly from the electronic medical record. We defined time to analgesia as time from patient placement in the treatment area (ED room or hallway) to time of analgesia administration, as documented in the electronic record. Analgesics were defined as any pain medication administered in the ED including "to go" medications, which almost always consisted of four tablets of acetaminophen with oxycodone handed to patients about the time of discharge. We defined opiate analgesia as any oral, intramuscular or intravenous medication administered in the ED as documented in the electronic record (e.g., acetaminophen with oxycodone, acetaminophen with codeine, morphine, hydromorphone). Non-opiate analgesia included any oral, intramuscular or intravenous non-narcotic medication (e.g., acetaminophen, ibuprofen, ketorolac). The main outcome measures were analgesic administration and time to analgesic treatment.

Data Analysis

Descriptive data are presented as means with standard deviations (SD) for continuous data, medians with interquartile ranges (IQR) for time variables, and frequencies and percents for categorical data. Patients were divided into

Table 1. Overall general patient characteristics

Characteristic	Overall n=24,752 (%)
Age [mean (years) ± SD]	42 ± 16.9
Age <65	21,950 (89)
Age 65-79	2,080 (8)
Age ≥80	722 (3)
Race	
African American	14,337 (69)
White	4,699 (23)
Other	1,690 (8)
Sex	
Female	16,047 (65)
Male	8,705 (35)
Presenting complaint	
Abdominal pain	18,804 (76)
Back pain	5,948 (24)
Pain score [mean ± SD]	7.5 ± 2.4
1-7	10,687 (43)
8-10	14,065 (57)
Triage classification	
1 (emergent)	347 (1)
2	7,103 (29)
3	15,036 (61)
4 (nonemergent)	2,257 (9)
Received any analgesia	15,128 (61)
Opiate	10,857 (44)
Non-opiate	4,271 (17)
Disposition	
Admission	6,124 (25)
Discharge	18,628 (75)

SD, standard deviation

three groups: age ≥80, age 65-79, and age <65. To assess differences in analgesia administration between age groups, we used the Fisher's exact test. We used the Kruskal-Wallis test to assess differences in time to analgesic administration between the groups. Times from room placement to analgesia are presented as medians with interquartile ranges (IQR). Potential confounding variables include gender, race, triage classification, severe pain, disposition status and presenting complaint of abdominal or back pain. We defined severe pain as a pain score of eight to ten. We determined this list of confounders *a priori,* and there were no stepwise techniques used to select variables. To calculate the likelihood of receiving analgesia or an opiate, while adjusting for the above confounders and clustering on physician, we used a generalized linear model with a log link, Gaussian error, and robust estimates of the standard errors of the model coefficients.[46] Data for these analyses are presented as relative risks (RR) with 95% confidence intervals (CI). We performed the generalized linear models using Stata statistical software (Version 11, Stata Corporation, College Station, TX). All other analyses were performed using SAS statistical software (Version 9.2, SAS Institute, Cary, NC). A probability of <0.05 was considered statistically significant.

RESULTS

We identified 24,752 patients with abdominal pain or back pain during the study period. See Table 1 for general patient characteristics. Overall, the group was young (mean age 42), more often female (65%) and black (69%), with a majority of patients presenting with abdominal pain (76%). The mean triage pain score for the group was 7.5. Overall, 15,128 patients (61%) received any analgesia, and 10,857 (44%) received an opiate analgesic.

The ≥80 years group (N=722; 3%), compared to the 65-79 years group (N=2,080; 8%) and to the <65 years group (N=21,950; 89%), was more often female (71 vs. 61 vs. 65%), black (72 vs. 65 vs. 69%), and had a lower mean pain score (6.6 vs. 7.1 vs. 7.6). Table 2 describes the age differences in patient characteristics. Both older groups were less likely to receive any analgesia (48 vs. 59 vs. 62%, p<0.0001), and the ≥80 years group less likely to receive opiates (35 vs. 47 vs. 44%, p<0.0001). Of those patients who received analgesia, both older groups waited significantly longer for their medication (median time 123 vs. 113 vs. 94 minutes; p<0.0001). After controlling for gender, race, triage classification, severe pain, disposition status, and presenting complaint, patients 65-79 were 8% less likely (95% CI: 6-10%) and patients ≥80 years were 17% less likely (95% CI: 14-20%) than the <65 years group to receive analgesia (Table 3).

DISCUSSION

The United States (U.S.) population aged 65 and over is expected to double in the next 25 years.[47] As the number of older persons continues to rise, emergency medical care will be increasingly affected. Oligoanalgesia, or the undertreatment of pain, is well documented in the EM literature and may disproportionately affect older adults.[2-4] Impairment in mental status has been shown to be highly prevalent among older ED patients with a lack of recognition of this impairment by emergency physicians.[48-51] Since 2000, The Joint Commission on Accreditation of Healthcare Organizations has made the assessment and management of pain a national quality standard and priority in the hospital accreditation process.[52] In addition, the EM community has also identified effective pain control as a quality metric.[17] Despite the significantly aging population, there is a paucity of data on the effect of age on analgesia use in the ED. We conducted this study to assess whether age affects the treatment of two of the most common chief complaints of patients seeking emergency care, abdominal pain and back pain.

Table 2. Age differences in patient characteristics

Characteristic	Age <65 n=21,950 (89%)	Age 65–79 n=2080 (8%)	Age ≥80 n=722 (3%)	p-value
Race				0.0002
African American	12717 (69)	1160 (65)	460 (72)	
White	4089 (22)	464 (26)	146 (23)	
Other	1498 (8)	157 (9)	35 (5)	
Sex				
Female	14,270 (65)	1261 (61)	516 (71)	<0.001
Male	7680 (35)	819 (39)	206 (29)	
Presenting complaint				0.54
Abdominal pain	16,662 (76)	1581 (76)	561 (78)	
Back pain	5,288 (24)	499 (24)	161 (22)	
Pain score [mean ± SD]	7.6 ± 2.3	7.1 ± 2.5	6.6 ± 2.6	<0.0001
1-7	9168 (42)	1087 (52)	432 (60)	
8-10	12,782 (58)	993 (48)	290 (40)	<0.0001
Triage classification				<0.0001
1 (emergent)	286 (1)	52 (3)	9 (1)	
2	6007 (27)	835 (40)	261 (36)	
3	13523 (62)	1084 (52)	429 (59)	
4 (nonemergent)	2125 (10)	109 (5)	23 (3)	
Received any analgesia	13,556 (62)	1,225 (59)	347 (48)	<0.0001
Opiate	9630 (44)	972 (47)	255 (35)	<0.0001
Non-opiate	3926 (18)	253 (12)	92 (13)	
None				
Disposition	8394 (38)	855 (41)	375 (52)	<0.0001
Admission	4846 (22)	910 (44)	368 (51)	
Discharge	17104 (78)	1170 (56)	354 (49)	

SD, standard deviation

Our study demonstrated older adults were significantly less likely to receive analgesia in a large cohort of more than 24,000 patients with abdominal or back pain. Our results are similar to prior studies of ED patients presenting with long bone fractures[12, 20] and in acute trauma.[35] As cognitive impairment is prevalent among older ED patients, this patient population may be at particularly high risk for oligoanalgesia. Older adults may present with significant pain and may not be able to advocate for their own care. We also found that of those patients who received analgesia, older patients waited significantly longer for their medication. Moreover, we found that this disparity was magnified in our oldest group of patients ≥80 years of age.

We identified several potential confounders in analgesia administration: gender, race, triage classification, severe pain, disposition status and presenting complaint of abdominal or back pain. In our study older adults were more likely to be female. A prior study of ED patients with acute abdominal pain demonstrated female sex was independently associated with being less likely to receive analgesic treatment.[5] However, older adults were still less likely to receive analgesia even after potential confounders, including gender, were adjusted for in our study. In our study, patients <65 years had a statistically significantly higher mean triage pain score (7.6) as compared to the older groups (7.1 in the 65 – 79 group and 6.5 in age ≥80 group). While this difference was statistically significant, we suggest that it is likely not clinically significant as providers should be motivated to relieve any degree of pain when pain is a component of the patient's reason for seeking emergency care. Most importantly, in our regression model, we adjusted for severe pain (a pain score of eight to ten) and older adults were still less likely to receive analgesia.

The literature on pain management in older adults suggests a variety of factors that may be obstacles to good pain control in this group. Patient-related factors include reluctance to report pain, fears of addiction with opiates, false belief that pain is a normal part of aging, and fear of being labeled a "bad" patient.[53-55] The patients in our study, however, not only reported their pain, but in fact rated it with a number on a pain score. Thus, it is more likely that the barriers in our

Table 3. Logistic regression of characteristics associated with analgesic treatment with clustering on physician

Analgesia		Relative Risk	95% Confidence Intervals		p-value
Any	White	1.03	1.01	1.04	0.001
	Female	1.03	1.01	1.04	<0.0001
	Pain score 8-10	1.20	1.18	1.22	<0.0001
	Triage classification	0.91	0.89	0.93	<0.0001
	CC of abdominal pain	0.78	0.75	0.80	<0.0001
	Admitted to hospital	1.29	1.27	1.32	<0.0001
	Age 65-79	0.92	0.90	0.94	<0.0001
	Age ≥80	0.83	0.80	0.86	<0.0001
Opiate	White	1.05	1.03	1.06	<0.0001
	Female	1.00	0.99	1.02	0.925
	Pain score 8-10	1.21	1.19	1.22	<0.0001
	Triage classification	0.90	0.88	0.92	<0.0001
	CC abdominal pain	0.95	0.93	0.98	<0.0001
	Admitted to hospital	1.35	1.33	1.37	<0.0001
	Age 65-79	0.97	0.95	0.99	0.002
	Age ≥80	0.86	0.83	0.89	<0.0001

CC, chief complaint

study were health-provider related. The reasons cited for poor performance at the clinician level include lack of effort to obtain an appropriate assessment, disbelief in patient's report of pain, exaggerated risk of adverse side effects, misinformation regarding addiction, and lack of training in treating elderly patients with pain.[18,56-59] Each one of these possible explanations deserves further attention clinically and more investigation. In addition to unnecessary pain and suffering, negative implications of oligoanalgesia in this patient population include depression and prolonged hospital length of stay.[31,32]

In older adults, an additional obstacle to effective analgesia is delirium, which can be exacerbated by both oligoanalgesia and overzealous analgesia.[30] The literature shows approximately 10% of older ED patients have delirium and an additional 16-22% suffer from cognitive impairment.[48, 50, 51] Patients who are cognitively impaired may not be able to advocate for their own care in the same way as other patients, which may include a decreased ability to communicate about pain or to request analgesia for painful conditions. While multiple tools exist to assess pain in cognitively impaired older adults,[60-63] most of these have not been studied in an emergency care setting.

Our study demonstrates a significant association between older age and decreased and delayed analgesia treatment in abdominal pain and back pain patients who presented to two urban EDs. These findings persisted after controlling for potential risk factors for oligoanalgesia. As the number of older adults continues to rise in the U.S. in the coming decades in the face of increasing use of emergency care, further research is critical to better understand and ameliorate this disparity, with the goal of providing the best possible pain management to older adults. The New Frontiers in Geriatric Research Task Force, in conjunction with the American Geriatrics Society, has previously defined a research agenda in geriatric care covering multiple specialties including EM.[64] The Task Force has advocated for improved residency training in the appropriate management of geriatric patients. A category Level B recommendation was assigned in support of prospective studies to assess the use of standardized protocols and educational models for improving the quality of care for older ED patients. We believe our findings support a need for further prospective investigations using standardized protocols to establish whether these interventions improve pain management in the geriatric ED population.

LIMITATIONS

Because this was a retrospective study we are limited in our ability to identify potential reasons (e.g. altered mental status, patient allergy) for delays or failures to initiate analgesic treatment in our population. Additionally, patient desire for pain medication, reassessment of pain scores, and discharge prescriptions for analgesia are unavailable for this analysis. As pain is a dynamic process, we are unable to measure if the evolution of pain influenced clinicians' willingness to prescribe analgesia. We did not assess other

potential agents used for analgesia in abdominal pain (e.g., viscous lidocaine, aluminum/magnesium hydroxide) or low back pain patients (e.g., muscle relaxants). As a nurse manually enters the time of medication administration into the electronic record, there may be a discrepancy between the recorded time and the actual time of administration. For the delay in analgesia, we defined time to analgesia as time from patient placement in the treatment area to time of analgesia, and thus it is unclear whether the delay was due to the ordering or administering of the analgesia, or both. Regardless, we found a delay in analgesia administration among older adults that requires further investigation with future research. We conducted our study in two urban academic centers, and thus our results may not be generalizable to other practice settings. In addition, we used a non-validated 4-point triage classification scale in this study. Other potential confounding variables in analgesia administration that were not available for our analyses include: hemodynamic instability, ED crowding, language barriers, health literacy, medication allergies, mental status or cognitive impairment impeding accurate assessment of pain, and physician bias.

Finally, it is possible that some of the decreased use of opiate medication in the older patients was due to appropriate caution taken by the prescribing physicians concerned about the changing pharmacodynamics of opiates in this population, making them more sensitive to both the analgesic and the adverse effects.[65] We would argue, however, that while this concern might have decreased the ordering of an opiate as a first pain medication, it should not have interfered with the use of any pain medication, especially the use of acetaminophen, which we included in our measure of analgesia. We also would have expected this concern to affect the dose of an opiate rather than the choice of an opiate. Furthermore, the delay in prescribing any pain medication in the older groups suggests that there may be other factors that either consciously or unconsciously result in less treatment of pain in older patients.

CONCLUSION

After controlling for potential confounders, older adults who presented to the ED for abdominal pain or back pain were less likely than younger adults to receive analgesia and waited longer for their medication.

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Older Emergency Department Drivers: Patterns, Behaviors, and Willingness to Enroll in a Safe Driver Program

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Objective: Our objective was to assess the reported driving patterns of older emergency department (ED) drivers and the factors that might lead them to enroll in a safe driving program.

Methods: We conducted a prospective, cross-sectional survey of a convenience sample of ED patients 65-years-old and up regarding their driving patterns, behaviors and willingness to enroll in a safe driving program.

Results: We surveyed 138 patients. Most (73%) reported driving within the last year, and 88% of these believe they could not manage without driving. Eleven percent of ED older drivers have been in a motor vehicle crash (MVC) in the past year (95% CI 6-20%), compared to 2.5% of all seniors. Our survey findings suggest that 88% of older ED drivers avoid at least some high-risk driving situations and 65% are unwilling to enroll in a safe driver program unless it lowers their automobile insurance rates. At the same time, most older ED drivers underestimate their risk of being involved in (75%) or dying from (74%) a MVC.

Conclusion: Overall, there are a significant number of older people for whom driving remains a vital yet risky daily function. Most of these drivers have little interest in information regarding safe driving programs while in the ED. Those willing to learn about such programs would prefer to take home the information regarding the program rather than have any staff member discuss it while in the ED. [West J Emerg Med. 2011;12(1):51-55.]

INTRODUCTION

The proportion of persons 65-years-old and over is increasing dramatically.[1] Automobile licensing rates will likely increase among the elderly.[2] Even without this impending demographic surge, there were already 30 million licensed older drivers in the U.S. in 2006. This represents an 18% increase from 1996, while the total number of licensed drivers increased by only 13% during the same time period.[3] Ninety percent of adults in the United States over age 65 rely upon private automobiles for transportation.[4] This declines to 75% of community-dwelling adults over age 75 who are thus at risk of becoming transportation disadvantaged. [5]

Older adult driving safety is a major public health issue. More stringent licensure requirements have not reduced fatalities.[6] Seventeen states have legislation requiring unique testing for older drivers.[7] The American Medical Association, the American Association of Retired Persons, and the National Highway Traffic Safety Administration have contributed programs, publications and guidelines to increase the safety and public awareness of older driving issues.[8,9,10]

Older drivers are at a higher risk of harming themselves and others compared to almost all other age groups of drivers even though they drive fewer annual miles.[11,12] Factors contributing to this risk include age-related declines in vision, such as reduced acuity, light sensitivity, peripheral vision capabilities, increased glare susceptibility, and slower ocular movements.[13,14] Cognitive skills allowing for rapid sequence tasking during driving compare unfavorably to younger drivers. [15] Also, short and long-term memory decline with age. Determinants of functional impairment such as reaction time, strength, and coordination diminish with age.[16] Prescription medications, such as benzodiazepines, antihistamines, antidepressants, analgesics and hypoglycemics, also contribute to increased crash risks.[17] Many of these factors impact older

drivers despite self-compensatory measures, such as cessation of nighttime and congested driving, driving fewer miles, avoiding passengers, and driving with a "co-pilot."[18-20]

Emergency physicians (EP) frequently evaluate many conditions that adversely affect the perceptual, cognitive and physical motor skills required for safe driving.[16,21,22] Emergency department (ED) visits may represent opportune moments for injury prevention education.[23] We undertook this study to gain a better understanding of older ED patients' driving patterns and problems and to explore their willingness to enroll in a safe driver program.[24]

METHODS

Study Design and Setting

We conducted a pilot, prospective, cross-sectional study in an urban, adult community teaching hospital ED (annual census 71,000). Approximately 23% of patients evaluated are aged 65 and older, and on average 35 older patients are seen in the ED daily between 7 AM and 11 PM. The hospital's Institutional Review Board approved this study.

Study Population

We included adult ED patients 65 and older who verbally consented to answer the survey. Research technicians were available to screen patients on weekdays from 7 AM until 11 PM. However, the sampling was not consecutive, as the technicians were involved in other concurrent research studies. We excluded patients in the ED for a motor vehicle crash (MVC), admitted from a nursing home, unable to communicate in English, who never drove before, and who were too ill to participate (e.g. altered level of consciousness, severe respiratory distress, myocardial infarctions, cerebrovascular accidents, intense active nausea and vomiting)

Study Protocol

We adapted the survey from other existing surveys and studies.[25,26] These questionnaires are believed to have face validity but have not been shown to have predictive ability for future MVCs.[26] We pre-tested our survey on five older ED patients for clarity, readability, and timing. Two research technicians administered the study. The survey queried older patients regarding their current driving patterns, perceived driving problems, and their willingness to enroll in a theoretical safe driver program. We recorded their preferences for a safe-driving program in terms of how they would prefer to obtain information, what information they would like to learn, and where they would like to attend a program.

Measurements and Data Analysis

We evaluated data with STATA®, version 8 (College Station, TX) and presented it as means and proportions with 95% confidence intervals. A confidence interval of +/- 10% for a single proportion could be obtained by enrolling 96 drivers.

Table 1. Characteristics of respondents

Driving Habits	Number (%) n=96
Frequency of driving	
Daily	53 (55%)
3-4 times per week	22 (23%)
1-2 times per week	18 (6%)
Less than 1 time per month	3 (3%)
Importance of driving	
Very important	84 (87%)
Somewhat important	8 (8%)
Not very important	4 (4%)
Miles driven per year	
Less than 6,000	55 (57%)
6,000-12,000	25 (26%)
More than 12,000	16 (16%)
Typical speed driven	
Less than limit	30 (31%)
At limit	58 (60%)
More than limit	8 (8%)

RESULTS

From September 2005 through January 2006, we approached 472 older ED patients. We excluded 241 patients, 50 were missed, and 43 refused. Reasons for exclusion included: too ill to participate (166), from a skilled nursing facility (48), in an MVC (5), non-English speaking (2), never drove before (19), and already enrolled (1). One hundred thirty-eight people agreed to participate. The mean age of those participating was 76 ± 6.4 standard deviation years and 51% were female. Out of these 138,101 reported having driven within the last one year (73%, 95%CI 65 – 80%), while 96 considered themselves current drivers (96/138 70% 95 CI 62-77%). Daily drivers made up 55% of current drivers (53/96 55% 95 CI 45-65%), 88% of current drivers stated they could not get along without driving (84/96 95% CI 79-94%), and 57% (55/96 95% CI 47-67%) drive somewhere between one and 6,000 miles per year (Table). Of the current drivers, 11% were involved in a crash within the past year (11/96 11% 95 CI 6-20%).

Most older drivers rated their own health compared to their peers (regardless of driving status) as either good or excellent (63/96 66% 96 CI 55-75%), while 89% (85/95 CI 81-95%) rated their driving compared to their peers as good or excellent. Three-fourths of those surveyed believe that compared to other groups, they are at the same or lower risk of both being involved in a MVC (71/95, 75%, 95% CI 65-83%) and dying in a MVC (70/94, 74%, 95% CI 64-83%) per mile driven. Older drivers reported sometimes avoiding high-risk

driving behaviors, such as driving at nighttime, on highways, in inclement weather, on heavily congested routes, and for long distances (Figure).

Of the 96 current drivers, 35 (36% 95 CI 27-47%) said that they would be willing to enroll in a safe driver program. However, 79% (76/96, 95% CI 70-87%) would be more interested in enrolling if it lowered their automobile insurance rates. Older drivers preferred taking home a video (53/96, 55%, 95% CI 45-65%) or a pamphlet (31/96, 32%, 95% CI 23-43%) to having their doctor (9/96, 9.3%, 95% CI 4.3-17%), nurse (0/96, 0%, 95% CI 0-3.8%), or a senior volunteer (3/96, 3.1%, 95% CI 0.6-8.9%) discuss driving safety with them while in the ED. The content older drivers most preferred for inclusion in safe driver programs included information on how to improve overall driving abilities (67/96 70% 95 CI 60-79%), information on how aging affects driving abilities (66/96 69%, 95 CI 58-78%), and how to lower their crash risk (54/96 56% 95 CI 46-66%). Less desirable topics included on-road lessons by driving instructors (32/96, 33% 95 CI 24-44%) and information on bad weather driving (35/96, 36% 95 CI 27-47%). Most subjects would prefer to attend a safe driving program at a local church or community center (55%, 53/96, 95% CI 45-65%), as opposed to a hospital facility (42%, 41/96, 95%CI 33-53%). The remaining participants would prefer a local driving school.

DISCUSSION

Many driving programs for older drivers already exist, yet no consistent improvement in lowering crash rates has been shown.[27-29] Prior to this study, the characteristics, behaviors and expectations of older drivers have not been established. Despite the fact that older drivers represent a high-risk age group for driving, the majority of older drivers do not express an understanding of this higher risk. Many of the ED drivers are frequent drivers, and it is considered very important to them. Our results concur with other surveys suggesting that older people do continue to drive using shorter trips, fewer miles, and self-imposed limitations to minimize risk, and they often describe visual and functional status limitations. [30]

In addition, older drivers who are in the ED for reasons unrelated to MVCs may represent a particularly high-risk group since they are involved in MVCs at a much higher rate (11% in the current study) than the general older population rate of 2.5%.[31] This may be evidence that an ED visit is a marker for increased frailty and functional dependence.

Despite the potential advantages, controversy remains surrounding the screening of older drivers and upon whom the burden of proof falls to demonstrate driving abilities.[32,33] Several resources are available for assessing older drivers.[8,34-36] These publications cover many issues pertinent to older drivers, including physical constraints, medical problems, cognitive status and dementia, medications, and the myriad of psychosocial aspects regarding driving cessation.

EPs face unique patient care challenges. Additional cognitive, physical, medica, and social screening may be impractical for a single ED visit. Even in primary care settings, barriers to effective screening include the potential liability for screening results, the lack of uniformity and predictive ability of existing screening methods, the possibility of patients reacting unfavorably to driving assessments, and a lack of resources.[37] Nonetheless, EPs must remain vigilant about the disposition of older ED drivers because nine states currently have relevant physician reporting laws.[7]

Overall, older drivers expressed little interest in attending a safe driver program except if it were to lower their automobile insurance rates. We were surprised that older drivers had such little interest in discussing safe driving issues with anyone while in the ED, including a senior volunteer.

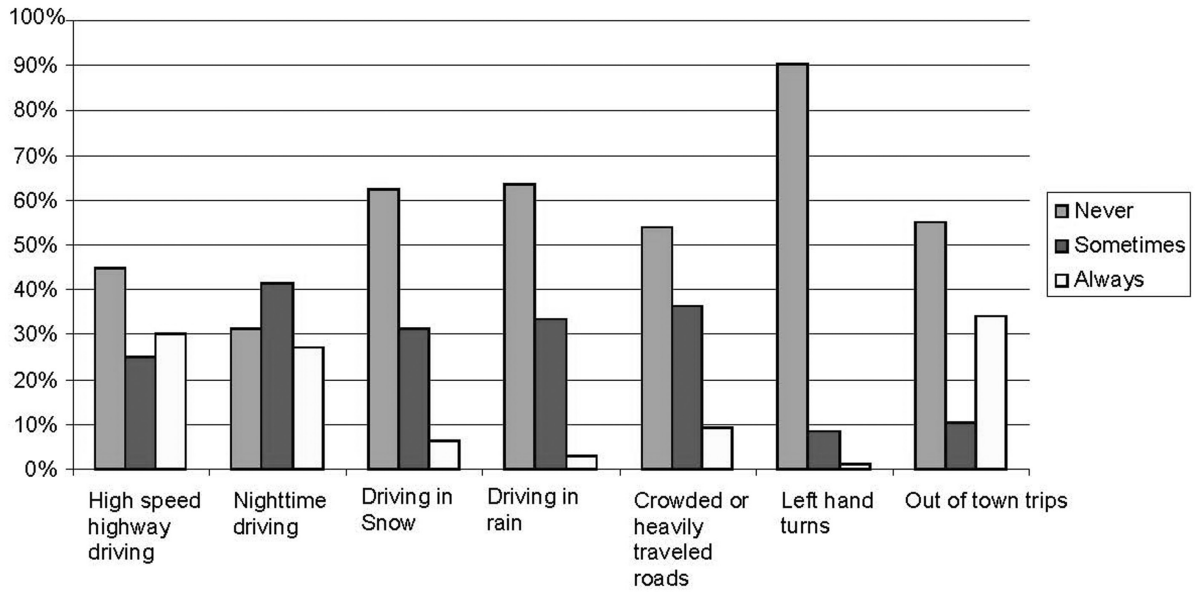

Figure. Avoidance behaviors by older ED drivers (n=96)

They did have a much greater willingness to learn via take-home materials. These issues should be taken into account when designing programs for intervention in this group. The fact that such a large percentage of the older drivers stated that they could not get along without driving may be due to the limited alternative means of transportation (such as carpooling, ridesharing, or public transit) within our community.

LIMITATIONS

There were no safe driver programs or materials offered to the study participants, therefore the impact of these on older drivers cannot be assessed. The survey has not been validated, nor been proven reliable. The nighttime exclusion, single site sampling of patients and the lack of a standard amount of alternative means of transportation may limit generalizability. In addition, neither the individual's functional status nor the familial influences on driving decisions were assessed. Lastly, typical times and reasons for driving, existing co-morbidities, and current medication use were not assessed.

CONCLUSION

Driving appears to be a very important aspect of daily living for many older ED patients. Most underestimate the increased risk of injury or death they incur from such activity compared to the remaining population. Several high-risk driving situations are often avoided by older drivers in an attempt to minimize difficulties. ED older drivers may represent a high-risk subgroup of older drivers. However, most still demonstrate little interest in a safe driving program, if approached while in an ED, unless it were to lower their automobile insurance rates.

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Cognitive Impairment among Older Adults in the Emergency Department

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Background: Within the next 30 years, the number of visits older adults will make to emergency departments (EDs) is expected to double from 16 million, or 14% of all visits, to 34 million and comprise nearly a quarter of all visits.

Objective: The objectives of this study were to determine prevalence rates of cognitive impairment among older adults in the ED and to identify associations, if any, between environmental factors unique to the ED and rates of cognitive impairment.

Methods: A cross-sectional observational study of adults 65 and older admitted to the ED of a large, urban, tertiary academic health center was conducted between September 2007 and May 2008. Patients were screened for cognitive impairment in orientation, recall and executive function using the Six-Item Screen (SIS) and the CLOX1, clock drawing task. Cognitive impairment among this ED population was assessed and both patient demographics and ED characteristics (crowding, triage time, location of assessment, triage class) were compared through adjusted generalized linear models.

Results: Forty-two percent (350/829) of elderly patients presented with deficits in orientation and recall as assessed by the SIS. An additional 36% of elderly patients with no impairment in orientation or recall had deficits in executive function as assessed by the CLOX1. In full model adjusted analyses patients were more likely to screen deficits in orientation and recall (SIS) if they were 85 years or older (Relative Risk [RR]=1.63, 95% Confidence Interval [95% CI]=1.3-2.07), black (RR=1.85, 95% CI=1.5-2.4) and male (RR=1.42, 95% CI=1.2-1.7). Only age was significantly associated with executive functioning deficits in the ED screened using the clock drawing task (CLOX1) (75-84 years: RR=1.35, 95% CI= 1.2-1.6; 85+ years: RR=1.69, 95% CI= 1.5-2.0).

Conclusion: These findings have several implications for patients seen in the ED. The SIS coupled with a clock drawing task (CLOX1) provide a rapid and simple method for assessing and documenting cognition when lengthier assessment tools are not feasible and add to the literature on the use of these tools in the ED. Further research on provider use of these tools and potential implication for quality improvement is needed. [West J Emerg Med. 2011; 12(1):56-62.]

INTRODUCTION

Within the next 30 years, the number of visits older adults will make to emergency departments (EDs) is expected to double from 16 million, or 14% of all visits, to 34 million and comprise nearly a quarter of all visits.[1,2] Compared to younger patients, older adults who visit the ED

are at increased risk for functional decline and medical complications, poorer management of pain and health-related quality of life and repeat ED visits.[3-7] With the aging of the population will come a surge in the number of older adults with cognitive impairment.[8-10] Approximately 26% to 40% of older adults who visit the ED have some form of cognitive impairment.[11-14] Among this group of patients found to be cognitively impaired in the ED, 80% have no prior history of dementia.[11,15] The Society of Academic Emergency Medicine (SAEM) Geriatric Task Force named cognitive assessment as one of the three major gaps in quality of care for geriatric patients.[16]

Long wait times, treatment in noisy and congested hallways, and the lack of daylight all may influence the onset and level of cognitive impairment when elderly patients present in the ED.[17] Because these exposures are more common during peak times, it seems logical that ED crowding may affect the presence of cognitive impairment in older adults.

Screening for cognitive impairment using long, detailed assessment scales, such as the Mini Mental State Examination (MMSE), is not practical in the ED because of limited time and competing demands. Alternatives include shorter scales, such as the Six-Item Screen (SIS).[18] The SIS is a brief scale that assesses orientation and recall. When compared to the MMSE, the SIS was found to be sensitive in detecting cognitive impairment in the ED (defined as MMSE<23); however, the sensitivity was lower than when the instrument was applied outside of the ED.[2] This is most likely due to the fact that the SIS only assesses temporal orientation (day, month, year) and recall (three items). Adding the clock-drawing task (CLOX) to test executive function offers a more comprehensive approach to screening for cognitive impairment. The CLOX1 is simple, easy to administer and has been used with older adults.[19-21] Knowledge of impairment in patients' orientation, recall or executive function is clinically important in the ED because deficits in one or more areas may impair patients' ability to provide an accurate medical history or medication list. Such deficits also negatively influence patients' ability to understand and follow discharge instructions.[2,22] To the best of our knowledge, no published studies have assessed using two tools (SIS and CLOX) to identify older adults with cognitive impairment in the ED.

The objectives of this study were to determine prevalence rates of cognitive impairment using the SIS and the CLOX1 and to identify associations, if any, between environmental factors unique to the ED and rates of cognitive impairment.

METHODS

Study Design

A cross-sectional, observational study of older adults admitted to the ED of a large, urban, tertiary academic health center was conducted to: identify rates of impairment among older adults; and identify relationships, if any, between ED environmental factors and presence of cognitive impairment.

Study Setting and Population

The ED at this academic health center contains 39 treatment beds with 25 private rooms and 14 hallway treatment areas. During the study period, the annual ED census was approximately 57,000 visits by adult patients, approximately 11.9% are >64 years of age.

Selection of Participants

Patients who presented to the ED between September 6, 2007, and May 1, 2008, were screened for cognitive impairment if they spoke English, were 65 years or older, lived within a 30-mile radius of the ED in the state of Pennsylvania, and lived independently (i.e., not in a nursing home). Patients were excluded from being screened for cognitive impairment if they had an end-stage disease with prognosis of six months or less, cancer diagnosis with active treatment, known alcohol or drug abuse, history of neurological disease (e.g., cerebral vascular accident with residual effects, multiple sclerosis, etc.), a previous medical history of dementia or delirium, or resided in a nursing home. These eligibility criteria and screening process presented here were established for a National Institutes of Health-funded large scale study (NIH/NIA R01-AG 023116).[23] These analyses are part of this larger patient screening effort for eligibility. The exclusion criteria were selected because these conditions are likely to have cognitive impairment that would already be known prior to our assessment. All patients who met the inclusion criteria and were present in the ED between 7AM and midnight, were approached by a trained research assistant in the ED who explained the screening and obtained verbal consent to be screened. This study was approved by Local Institutional Review Board.

Methods of Measurement

We assessed cognitive impairment using two validated screening tools: the SIS and CLOX1.[2,18,19] SIS is a brief and reliable scale designed to identify subjects with deficits in orientation and recall.[18] The patient is asked three temporal orientation questions (day of the week, month, year) and three recall items (hat, car, tree). Each correct answer is given a point towards a summed score (range: 0-6). The SIS has been used in the ED and with older adults as a screen to identify cognitive impairment among potential older adult research subjects.[2,18] Patients with greater than two errors on the SIS were considered impaired. Patients who made fewer than two errors on the SIS were asked to complete a CLOX.The CLOX1 was chosen to assess for executive function impairments. Scores range from 0-15 in this subscale of the larger CLOX,[19] with scores ≤10 indicating deficits in executive functioning. The CLOX1 tests executive control function and is strongly associated with cognitive test scores.[20] The executive control

functions are cognitive processes that coordinate simple ideas and actions into complex goal-directed behaviors. Examples include goal selection, motor planning sequencing, selective attention, and the self-monitoring of one's current action plan. All are required for successful clock drawing. Together the SIS and CLOX1 take under five minutes to complete, and each is associated with severity of cognitive impairment.[18,19] Patients were considered to have cognitive impairment deficits if they scored ≤4 on the SIS or ≤10 on the CLOX1.

Several ED specific environmental variables were documented by research assistants on a standardized data collection form. Patient triage time, "in-room" (or hallway) time, total patient-care hours (a sum of all the hours for all patients currently in the ED), number of admitted patients (number of patients who are admitted to the hospital but currently boarding in the ED), waiting room number (number of patients in waiting room) and triage level were queried from EMTRAC (Hospital of the University of Pennsylvania, Philadelphia, PA), a computerized patient tracking and charting system, and recorded in real time on each study form. We calculated total minutes waiting in triage by subtracting triage time from "in-room" time for each patient. Triage level is a nurse-assessed four-point scale (1-4) based on the urgency of the ED patient's complaint, where "1" signified most emergent cases and "4" signified least urgent. Patient demographic information (race, gender, age), location of screening (hallway or private room), and if the patient was admitted or discharged to the hospital were also collected for each patient.

Outcome measures

There were two primary outcomes for this study. The first was to assess the prevalence of cognitive impairment among older adults visiting the ED, using the SIS and CLOX1. The second was to examine the relationship between cognitive impairment assessment (screened positive for cognitive impairment on either SIS or CLOX1 versus no impairment) and various patient and ED characteristics and to identify which environmental factors, if any, are associated with the assessment of cognitive impairment in the ED for older adults.

Primary Data Analysis

Data are reported as frequencies and percentages for categorical data, and as means ± standard deviation and range for continuous variables. Among patients with multiple ED visits during the study period, only data from one of the patients' visits were used in our analyses.

We used a generalized linear model with a log link, Gaussian error, and robust estimates of the standard errors of the model coefficients to calculate relative risk (RR). We controlled for several patient-specific characteristics (age, sex, race [black], whether the patient was admitted to the hospital, time of day triaged at ED [7AM-3PM, 3PM-11PM, 11PM-7AM]), and ED characteristics (triage class [as an indicator variable compared to the most severe triage score], waiting time, crowding, location of interview [private room]) in a final model. We determined the list of confounders *a priori*. To control for the affect of all *a priori* confounders all were included in a final model; no stepwise techniques were used to select variables. Given the large number of outcomes, the study was adequately powered for multivariable analysis. We used the Bonferroni correction of n=14 to adjust for covariates because of the multiple statistical tests performed on the data. Based on this, a probability of <0.003 was considered statistically significant.

Data for these analyses are presented as relative risks

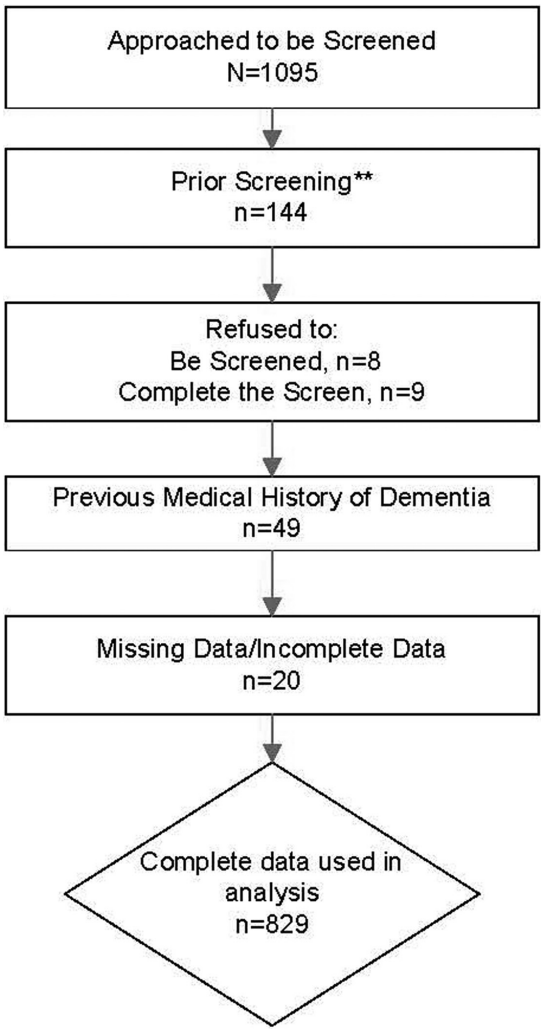

Figure 1. Patient participation
** There were 126 unique patients screened a total of 144 times; 114 patients had one additional screen, nine patients had two additional, one patient had three additional screens, one patient had four additional screens, and one patient had six additional screens.

with 95% confidence intervals. We performed all analyses using Stata statistical software (Version 10, Stata Corporation, College Station, TX).

RESULTS

Characteristics of Study Subjects

Between September 6, 2007, and May 1, 2008, 1,095 patients were admitted to the study and were approached by the research assistants, of whom 266 were subsequently excluded (Figure 1). Of the remaining 829, patients were predominantly black (67.5%) and female (65.1%). Patients ranged from 65 to 105 years old (mean age: 75.7± 7.1 standard deviation). See Table 1.

Table 1. Characteristics of emergency department patients screened for cognitive impairment (N=829)

	Mean±SD(range)
Age, *years* mean±sd (range)	75.7 ± 7.1(65-105)
	n(%)
65-74 years	405(48.9%)
75-84 years	322(38.8%)
85+ years	102(12.3%)
Gender	
Female	539(65.0%)
Male	290(35.0%)
Race	n(%)
African American, n(%)	561(67.7%)
Caucasian, n(%)	243(29.3%)
Other, n(%)	16 (1.9%)
Unknown, n(%)	9 (1.1%)
Time of day Triaged	n(%)
7 AM-3 PM	457(55.1%)
3 PM-11 PM	327(39.5%)
11 PM-7 AM	45(5.4%)
Triage level, *1=emergent 4=non-urgent*	
1	26(3.1%)
2	510(61.5%)
3	272(32.8%)
4	21(2.5%)
	Mean±SD(range)
Number of people in waiting room	13.5±9.1 (0-42)
Number admitted patients in emergency department	8.4±6.5 (0-50)
Total patient hours, *minutes*	181.8±88.3 (19-579)
	n(%)
Screened in private room	667(80.5%)
Admitted	438(52.8%)

Note: Due to rounding percentages may not add to 100%.
SD, standard deviation

The prevalence of cognitive impairment assessed using the SIS and CLOX1 is summarized in Table 2. A total of 42% percent (350/829) presented with deficits in orientation and recall. An additional 36% (297/829; 62% of patients [297/479] visiting the ED who passed the SIS) had deficits in executive function. None of these patients had a documented history of dementia or other cognitive impairment noted at the ED visit.

Table 3 shows the results from the multivariate analysis. The adjusted analyses for screening positive using the SIS and the subsample that were screened using the CLOX1 showed that only patient demographics were significantly associated with screening positive for cognitive impairment in the ED. Specifically patients were more likely to screen positive for cognitive impairment using the SIS if they were 85 years or older (RR 1.63, $p<0.001$), black (RR 1.85, $p<0.001$) and male (RR 1.42, $p<0.001$). Interestingly, only age was significantly associated with screening positive for cognitive impairment in the ED using the CLOX1 (75-84 years: RR 1.35, $p<0.001$; 85+ years: RR 1.69, $p<0.001$). Race and gender were no longer significant in the full model when using the CLOX1 to assess for deficits in executive functioning with this clock-drawing task. Time of day triaged, number of people in the waiting room, number of admitted patients, total patient hours, being screened in a private room, and the admission status of the patient were not associated with screening positive for cognitive impairment in the ED in either of the full models.

DISCUSSION

This study explored whether the SIS and CLOX1, executive clock-drawing task, can be used in the ED to easily

Table 2. Six-Item Screen (SIS) and clock-drawing task (CLOX1) scores

Screen	Mean±SD(range)
Six-item Screen, N=829	4.5±1.5 (0-6)
Score:	n(%)
0-2	74(8.9%)
3-4	276(33.3%)
5-6	479(57.8%)
	Mean±SD(range)
CLOX1, N=479**	8.9±3.4 (0-15)
Score:	n(%)
0-5	90(18.9%)
6-10	207(43.2%)
11-15	182(38.0%)

Note: Due to rounding percentages may not add to 100%.
** N=479 because patients who scored 4 or below on the SIS indicating deficits in orientation and/or recall were not screened with the CLOX1 clock drawing task as was part of the recruitment criteria for the larger study.
SD, standard deviation

Table 3. Patient and emergency department characteristics associated with screening positive for cognitive impairment

Characteristic	SIS (N=829) RR (95% CI)	CLOX1 (N=479) RR (95% CI)
Age:		
65-74 years (*ref group*)	-	-
75-84 years	1.32(1.08-1.60)*	1.35 (1.16-1.57)**
85+ years	1.63 (1.28-2.07)**	1.70 (1.46-1.96)**
Race, *African American*	1.85 (1.45-2.37)**	1.06 (0.92-1.22)
Gender, *Male*	1.42 (1.20-1.68)**	1.11 (0.97-1.28)
Triage time:		
7 AM – 3 PM (*ref group*)	-	-
3 PM – 11 PM	1.18 (0.99 -1.40)	1.10 (0.96-1.25)
11 PM – 7 AM	1.10 (0.71-1.70)	1.13 (0.90-1.41)
Class:		
1 (*ref group*)	-	-
2	1.06 (0.61-1.82)	1.19 (0.71-1.99)
3	1.02 (0.59-1.77)	1.30 (0.77-2.18)
4	0.65 (0.23-1.92)	0.25 (0.06-1.10)
Waiting room number	1.00 (0.99-1.02)	1.01 (1.00-1.02)
Number of admitted patients in emergency department	0.99 (0.98-1.01)	1.00 (0.99-1.01)
Total patient hours	1.00 (0.998-1.00)	1.00 (1.00-1.00)
Screened in private room	0.92 (0.74-1.15)	0.96 (0.81 – 1.13)
Admitted	1.04 (0.87-1.26)	1.06 (0.92-1.22)

* Significant at p=0.006. ** Significant at p < 0.001.
SIS, Six Item Screen; *CLOX1*, executive clock drawing task; *RR*, relative risk; *CI*, confidence interval.

assess and identify possible cognitive impairment among older adults. A high proportion of patients presented to the ED with deficits in orientation and recall (42%) and among those elders who passed the SIS, 36% had executive-functioning deficits. Only demographics (age, race, and gender) were significantly associated with screening positive for cognitive impairment in the ED. None of the environmental factors assessed were significant in the adjusted model. These results have four major implications.

First, a substantial number patients age 65 and older admitted to the ED present with impairments in orientation, recall and/or executive functioning. Our findings with respect to deficits in orientation and recall are consistent with those other studies that have reported between 26% and 40% of all ED elders have some form of cognitive impairment.[2,15] These studies included people with dementia as well as delirium. This tool is short and easy to administer, allowing for a quick and reliable way of assessing deficits in orientation and recall, which may affect a patient's ability to provide reliable self-report of symptoms and medications, as well as influence a patient's ability to remember instructions post discharge from the ED. With respect to the assessment of executive function, there has been only one study to our knowledge that has used CLOX1 as part of a cognitive assessment in the ED.[14] The tool used in that study did not score this task in as much detail to determine the level of impairment in executive functioning and was part of a tool that had a three-item recall (Mini-Cog).[24] The clock-drawing task identified an additional 36% of older patients seen who have deficits in executive function with few or no deficits in orientation and recall (SIS >4). These results highlight the fact that although a similar proportion of older adults being seen in the ED have deficits in orientation and recall, a subset of older adults being seen in the ED have significant deficits in executive function. Together these two tools can be used to assess and document cognitive impairment in older adults in the ED, as was recommended by the SAEM Geriatric Task Force as a quality indicator.[16] Using both tools can provide a more comprehensive snapshot of the patient's cognitive status at admission to the ED.

Second, being male in our sample was significantly associated with positive screens for cognitive impairment using the SIS. This is an interesting finding since women are more likely than men to have Alzheimer's disease or other types of dementia.[8] However, this is due to women living longer.[25] Yet in our data, after controlling for age and race, being male was still a significant predictor to screening positive for cognitive impairment in the ED using the SIS. The age group 75-84 years was not a significant predictor of screening positive on the SIS based on our Bonferroni correction ($p \leq 0.003$). However, it is worth noting that the p-value was 0.006 and rejecting this as a significant predictor may be a Type II error. Future research assessing gender differences using the SIS as a screening tool for research subjects in the ED is needed.

Third, being black in this sample was also a predictor of screening positive for cognitive impairment using the SIS. Other researchers have found that racial differences do not persist when age, gender, education and comorbid conditions are included in the analyses.[25-28] It is possible that comorbid conditions and educational level may confound this association, but we did not collect that data.

Finally, this is the first study known by the authors to have controlled for no ED-specific environmental variables (e.g., crowding, time of triage, triage class, location of screening, wait time, etc.) in relation to screening cognitive impairment. The finding that only patient demographics were significant predictors of cognitive impairment while no ED-specific environmental variables supports the use of assessment tools such as the SIS and CLOX1 in the ED. Future research over the length of an ED stay to confirm that these environmental factors are not associated with the assessment of cognitive impairment is needed.

LIMITATIONS

There are a number of limitations to this study. First, we did not assess changes in impairment over time, which may occur with prolonged ED stays or though progression of disease. We found no association between ED crowding and cognitive impairment, which may be because patients were approached early in their ED visit and may not have had sufficient exposure to be injured by the crowded environment.

Second, screening of patients was limited to 7AM to midnight; therefore, elderly patients triaged between midnight and 7AM were missed. However this is <10% of this population that present to the ED during midnight to 7AM. Third, delirium was not formally assessed using a separate tool such as the Confusion Assessment Method.[29] It is possible that some elders who screened positive on either scale were in fact experiencing delirium at the time of the screening. Other researchers have reported that approximately 7-10% of patients screen positive for delirium in the ED.[11,15] However, the research previously published using the SIS in the ED did not differentiate delirium and other types of cognitive impairment stating, "A hallmark of the diagnosis of delirium is recognizing that impairment of memory and orientation exists."[14,22] Overall, the use of assessment tools, such as the SIS and CLOX1, support the SAEM Geriatric Taskforce recommendation that rapid and objective assessment and documentation of cognition in older adults in the ED as a quality indicator.

Finally, we were limited in that the assessment was only conducted in the ED. Impairment may develop or recede later if patients are screened after admission to the hospital. Future studies should include a follow-up assessment of cognitive impairment to determine changes over time and specific assessment for other types of impairments, such as delirium. Finally, the generalizability of the results may be limited due to this study being conducted at only one academic urban ED and the high proportion of black study members.

CONCLUSION

These findings suggest that a high proportion of elders are either admitted to the hospital from the ED or are being discharged from the hospital with cognitive deficits, specifically loss of executive function. The SIS assessment of orientation and recall coupled with a clock-drawing task can be used in the ED to easily assess cognition. These two tools (SIS and CLOX1) provide a rapid and simple method for assessing and documenting cognition when lengthier assessment tools are not feasible and add to the literature on the use of these tools in the ED. Further research assessing the impact of using these brief cognitive screens on the course of treatment (i.e., in ED, during hospitalization, and post discharge follow up assessments) and potential implication for quality improvement is needed.

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Urine Test Strips to Exclude Cerebral Spinal Fluid Blood

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Introduction: Determining the presence or absence of red blood cells (RBC) or their breakdown products in cerebrospinal fluid (CSF) is essential for the evaluation of subarachnoid hemorrhage (SAH) in headache patients. Current methodology for finding blood in the CSF is either spectrophotometric detection of pigment, which is time consuming and labor intensive, or visual assesment of samples for color change (xanthochromia), which is inaccurate. Bayer Multistix® urine test strips are designed to test urine for RBC by detecting the presence of hemoglobin. The aim of this pilot study was to evaluate the perfomance of urine reagent test strips for ruling out the presence of RBC in CSF.

Methods: We compared color changes on Multistix® urine test strips to the standard of spectrophotometric absorbtion at 415nm and initial RBC counts in 138 visually clear CSF samples.

Results: We performed Pearson Chi-Square and likelihood ratios on the results and found a correlation between a negative result on the urine test strip and less than 5 RBC per high power field and a spectrophotometric absorbance of less than 0.02% at 415nm in a CSF sample.

Conclusion: These results warrant further investigation in the form of a prospective clinical validation as it may alter the emergency department evaluation for SAH. [West J Emerg Med. 2011;12(1):63-66.]

INTRODUCTION

Patients presenting to the emergency department complaining of rapid onset or "thunderclap" headache require an urgent clinical evaluation to rule out a subarachnoid hemorrhage (SAH). This process typically involves a non-contrast head computed tomography and, if negative, a lumbar puncture to determine the presence of blood or blood products in the cerebrospinal fluid (CSF). The characteristic, faint yellow staining of CSF by RBC breakdown products is known as xanthochromia. When red blood cells (RBC) lyse in CSF they release hemoglobin that is later enzymatically converted to methemoglobin and bilirubin. The number of intact blood cells is typically determined by either an automated cell counter or by counting cells per high power field in manual microscopy. Most United States laboratories currently determine xanthochromia by using a visual testing method in which a sample of CSF is compared to a white background and any staining present is noted. Unfortunately results can be highly inaccurate if the visual testing method is employed.[1] The more accurate but less common method for determining xanthochromia is to use a spectrophotometer and record a sample absorbance at wavelengths known to correspond with breakdown pigments, such as hemoglobin, methemoglobin and bilirubin. Laboratory evaluation of CSF can require one to two hours or more to complete if spectrophotometric methods are used. Quickly and reliably confirming the lack of clinically significant levels of RBC in CSF might significantly expedite the evaluation of these headache patients. When evaluating a patient for the possibility of SAH a negative reagent test for blood might obviate further lab investigation or imaging. A straightforward, rapid reagent test has the potential to save clinical time and diagnostic costs.

Other medical settings in which reliable, expeditious CSF results would be valuable are those with less access to

Table 1. Absorbance at 415nm.

		Absorbance at 415nm		
		>= 0.021	< 0.020	Total
Multistix® Result	positive	32	43	75
	negative	3	60	63
Total		35	103	138

Table 2. Red blood cells per high power field.

		Red blood cells per high power field			
		>10	5-10	0-5	Total
Multistix® Result	positive	26	12	37	75
	negative	1	0	62	63
Total		27	12	99	138

technology and resources. Across Third World nations and in military Combat Support Hospitals, patients requiring evaluations for headaches may not have access to radiology, photo spectrometry, microscopy or automated cell counters. In these austere settings, visually ruling out xanthrochromia becomes paramount. A reagent test that capitalizes on simplicity and economy would better serve these resource-constrained facilities.

METHODS

Research data were derived as an approved study under the supervision of the local Institutional Review Board/ Institutional Animal Care and Use Committee protocol (CIP# 2006.0027). We conducted a laboratory evaluation from July to October 2006, using CSF samples submitted to the hospital laboratory. In the vast majority of cases the volume of CSF submitted to a lab is more than is needed for the requested diagnostic evaluation. This study intercepted this surplus CSF prior to disposal. The CSF was stored for up to one week at 4°C until analyzed, then allowed to warm to room temperature prior to testing. We inspected all samples to eliminate those with any visually discernible xanthochromia. This visual sorting was undertaken to narrow our test pool only to samples that would have been classified as negative using the common practice methods in place in most labs. A visually pigmented sample needs no further evaluation as it is already positive. The investigators found 138 samples to be visually clear.

We placed each clear sample placed in a Bausch & Lomb™ Spectronic 601 spectrophotometer and recorded the percent light absorption at 415 nm. Xanthochromia is considered present in CSF when spectrophotometric absorbance ≥0.024% at 415nm. Graves et al[2] has recently suggested a more conservative value of ≥0.023%. We used a cutoff of ≥0.02% at 415nm to provide a safety margin and to reflect the trend toward a more stringent threshold for xanthochromia.

A different investigator used a Bayer Multistix® urine test strip to test the same sample for the presence of blood. Following the manufacturer's procedure for urine testing, we placed a drop of sample CSF on the blood square on the urine test strip. At 60 seconds, we noted and compared the color change against the key found on the side of the container. Since the CSF used in this study were samples that had already been analyzed by the hospital lab, at the end of each testing session we looked up and recorded each sample's cell counts after visual sorting, spectrophotometry and urine test strip analysis was completed. We then compared the urine dipstick testing results to the CSF blood cell counts and spectrophotometric results for statistical analysis.

We maintained a pooled sample of CSF known to be free of red cells and tested it biweekly with urine strips to ensure that the test strips would not yield false positive results due to aging CSF or other unanticipated factors. This true negative standard was made from CSF samples that had shown no absorbance on spectrophotometry, had tested negative on the test strips, and did not contain greater than five red cells per high power field. We used this standard as a measure to ensure that true negative results on the test strips were reliable, reproducible, and durable.

RESULTS

Since the goal of this project was to use urine test strips to rule out the presence of red cells in CSF, we distributed the results into two groups. Group one consisted of samples with negative urine dip results. Group two was comprised of samples showing any positive dip result. We considered a sample positive if there was any color change on the test strip, regardless of the magnitude or pattern of the color change. Sixty-three of 138 samples tested negative for blood using the urine dipstick method.

We assigned spectrophotometer results above the cutoff to one group, below to a second. Using spectrophotometric methods, we found that 103 of 138 samples tested negative for xanthochromia.

The microscopic analysis results for RBCs per high power field were assigned to one of three groups: 0-5, 6-10, and >10. We chose two cutoffs for red blood cells (5/hpf and 10/hpf) to represent common lab thresholds for "normal" red cells in CSF. Ninety-nine samples had ≤5 rbc/hpf, 12 samples had 5-10 rbc/hpf, and 27 samples were >10 rbc/hpf. The Multistix® proved to be very sensitive. Of samples with zero to five cells per high power field, 37 showed some positive results. When a threshold of zero cells per high power field was used, 15 out of 138 samples were false positive.

We conducted two Pearson Chi-Square tests. The first compared samples above and below the spectrophotometric

Table 3. Absorbance vs. Multistix®

	Absorbance vs. Multistix®	
	Value	Asymp Sig (p-value)
Pearson Chi-Square	25.987	0.000
Continuity Correction (2x2)	24.024	0.000

Table 4. Red blood cells per high power field vs. Multistix®

	Red blood cells per high power field vs. Multistix®	
	Value	Asymp Sig (p-value)
Pearson Chi-Square	40.726	0.000

cutoff to samples with negative and non-negative results on the urine test strips (Table 1). The second compared the three sets of red cell results to negative and non-negative results on the urine test strips (Table 2). We found significant correlation ($p<0.0001$) between a negative result on the urine test strip and the negative cutoff of 0.020% spectrophotometric absorption at 415nm. We found this same correlation ($P<0.0001$) between a negative test strip result and a CSF microscopic reading <5 RBC/HPF. Tables 3 and 4 depict the statistical results.

When compared to spectrophotometry, the Multistix® had a sensitivity of 0.91 and a negative predictive value of 0.95, with a specificity of only 0.58. The negative likelihood ratio was 0.16. When compared to the RBC/HPF (cutoff of ≤5), the Multistix® had a sensitivity of 0.97, a negative predictive value of 0.98, with a specificity of 0.62. The negative likelihood ratio was 0.04. We used the highly conservative spectrophotometric cutoff value of 0.020 for this analysis; only further work will determine if this cutoff was too conservative.

The true negative pooled CSF yielded no false positive reaction on the Multistix® during the four-month period of this study.

DISCUSSION

A SAH occurs with the release of blood into the CSF. It subsequently breaks down over time into the characteristic pigments of hemoglobin, bilirubin and methhemoglobin. Hemoglobin from red cell lysis is the first significant compound, presenting as early as two hours, peaking at around 24 hours, and persisting for up to a week. Bilirubin is only created in vivo and much later than hemoglobin, as its creation requires the action of an arachnoid oxygenase not present in the test tube once a sample is collected, making it much more difficult to study. Methemoglobin represents another RBC breakdown product that, like bilirubin, is only encountered after time. Typically patients with severe headaches present acutely, often within a few hours of onset of their symptoms. This makes hemoglobin the best target pigment for this pilot work in test strip diagnosis of blood in the CSF. As Multistix® include a Bilirubin test block, subsequent clinical exploration should include this pigment.

We were concerned about the number of samples that were visually pigment free, per the inclusion criteria for this study, but which subsequent microscopic or spectrophotometric analysis indicated were positive for blood. These findings support other authors'[1, 3, 4] misgivings about using visually determined xanthochromic absence to rule out intrathecal blood. Unfortunately, testing for xanthochromia using visual methodology is still employed by many hospitals, particularly in the United States. Should urine test strip results correlate well with spectrophotometric results, they would provide for a quick, easy alternative to visual xanthochromia determination in centers without access to spectrophotometery.

An examination of the data reveals almost two thirds of the visually clear samples tested negative for blood via reagent strips. Hence the statistical reassignment was undertaken. Our goal was to evaluate the performance of the Multistix® in a Boolean, yes/no paradigm. In other words, we wanted to know how good a negative result on the Multistix® was. When compared to the gold standard of spectrophotometery for xanthochromia, the Multistix® showed promise as a simple test to rule out red cells in the CSF. SAH is a life-threatening condition. High test sensitivity is essential in a screening test for detecting of intrathecal blood. With a high sensitivity a resultant increase in false positive results is to be expected. The false positive rate would need to be closely monitored if this diagnostic method were to be evaluated in a clinical setting. It may subsequently be shown that CSF testing with urine test strips is only useful when the result is negative.

As the focus of this study was a bench-top evaluation of the performance of urine test strips in analyzing CSF, the circumstances and settings under which we obtained these samples were of secondary importance. Often novel diagnostic strategies are evaluated directly in a clinical setting without first determining whether they perform in a controlled laboratory setting.

Our use of an all comers convenience sample implies that some of the CSF we analyzed was obtained from patients in whom other diagnoses, such as infection rather than subarachnoid hemorrhage, were being entertained. Might there be some protein or entity present during an infection and not present during simple hemorrhage that would yield a false positive result? Bayer assured the investigators that the "heme" portion of the Multistix® was specific for blood. Unfortunately the exact mechanism of the colorimetric reaction is understandably proprietary. Multistix® detect hemoglobin in the cell; therefore, it can be inferred that the process of spotting samples onto urine reagent strips lyses

red cells to release their contents for subsequent detection by colorimetric chemical reaction. This lysis effectively removes the potential confound of early tap analyzed prior to cell lysis and release of pigment.

LIMITATIONS

This is a relatively small, bench-top pilot study; no clinical implications are suggested or warranted without proper prospective validation.

Traumatic tap rates are reported as high as 10%.[3] As urine test strips most likely lyse cells in their detection of pigment, they would be unlikely to assist with detecting a traumatic tap caused by the presence of hemoglobin. Bilirubin theoretically shows more promise for differentiating traumatic lumbar puncture from subarachnoid hemorrhage, with the important caveat that it would only be expected to be present at 12 hours after the onset of bleeding.[4] As this study used lab surplus CSF samples of varying ages, we did not assess the performance of the Bilirubin panel on the urine test strips.

The use of surplus samples made it impossible to control for the clinical setting of the lumbar puncture. These samples left us unable to ascertain the indications for the procedure or the technique employed to obtain the samples. Some authors have suggested that using Betadine® as a skin prep can introduce spurious coloration to a sample.[5] While it is unlikely that storage of CSF affected the results, this study's use of lab samples represents a potential confound that can only be addressed by a clinical trail.

CONCLUSION

Given a correlation between a negative result on the urine test strip and less than five RBC per high power field and a spectrophotometric absorbance of less than 0.02% at 415nm in a CSF sample, determining the absence of CSF RBCs via benchtop CSF evaluation of urine test strips shows enough promise to warrant further study in a prospective clinical setting. Ultimately this may prove to be an expeditious and cost-effective adjunct to the evaluation of headaches when ruling out subarachnoid hemorrhage.

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Disclosures: The authors have no financial or proprietary interests to disclose. I am an employee of the U.S. government. This work was prepared as part of my official duties. Title 17 U.S.C. 105 provides that 'Copyright protection under this title is not available for any work of the United States Government.' Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties. Research data derived from *Ruling out CS blood using urine reagent testing strips*, an approved Naval Medical Center, Portsmouth, VA IRB/IACUC protocol (CIP#2006.0027).

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Imaging in Acute Stroke

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Imaging in the acute setting of suspected stroke is an important topic to all emergency physicians, neurologists, neurosurgeons and neuroradiologist. When it comes to imaging, the American College of Radiology (ACR) continually updates its guidelines for imaging pathways through the ACR Appropriateness Criteria.[1,2] This article is a general review of the imaging modalities currently used to assess and help guide the treatment of strokes. [West J Emerg Med. 2011;12(1):67-76.]

NONCONTRAST COMPUTED TOMOGRAPHY

Rapid evaluation of acute stroke patients will increase as the population ages and acute therapies expand. One significant aspect in the evaluation of acute ischemic stroke patients is imaging. Currently in the United States, noncontrast computed tomography (CT) remains the primary imaging modality for the initial evaluation of patients with suspected stroke (Figure 1).[1,2]

Three main stages are used to describe the CT manifestations of stroke: acute (less than 24 hours), subacute (24 hours to 5 days) and chronic (weeks).[3] Acute stroke represents cytotoxic edema, and the changes can be subtle but are significant. They are also termed "early ischemic changes "and were formerly termed "hyper-acute". It is intracellular edema and causes loss of the normal gray matter/white matter interface (differentiation) and effacement of the cortical sulci. A thrombus in the proximal middle cerebral artery (MCA) is sometimes seen in the acute phase and appears as hyperattenuation. A subacute stroke represents vasogenic edema, with greater mass effect, hypoattenuation and well-defined margins. Mass effect and risk of herniation is greatest at this stage. Chronic strokes have loss of brain tissue and are hypoattenuating.

A noncontrast head CT may identify the early signs of stroke, but most importantly will exclude intracerebral hemorrhage and lesions that might mimic acute ischemic stroke such as tumor or intracerebral hemorrhage. Noncontrast CT is also used in the evaluation of acute intracranial hemorrhage as it produces good contrast between the high attenuating ("bright") clot and the low attenuating ("dark") cerebrospinal fluid (CSF) [Figures 2-4].[4] This tool's availability and speed make it very useful in the initial evaluation of suspected stroke patients.

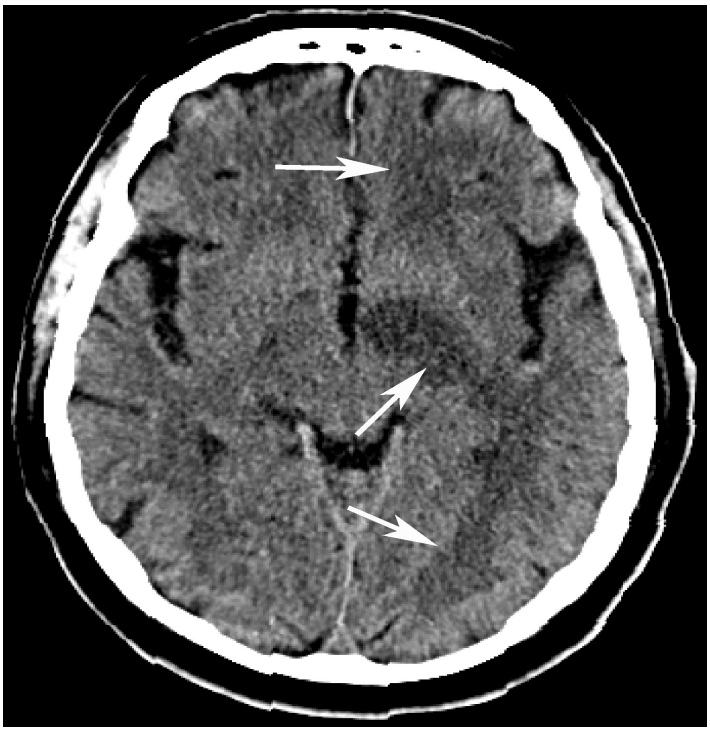

Figure 1. Left middle cerebral artery (MCA) infarction. Axial nonenhanced computer tomography demonstrates hypoattenuating foci throughout the left sided white matter (arrows) and sulcal effacement in the left MCA territory, consistent with infarction.

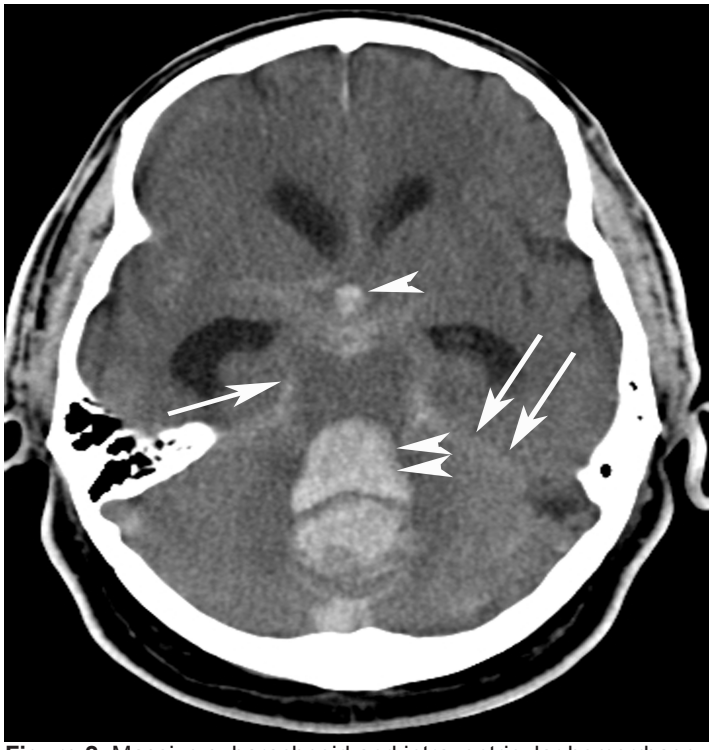

Figure 2. Massive subarachnoid and intraventricular hemorrhage**.** Axial nonenhanced computer tomography demonstrates a large" bright" or hyper attenuating dense subarachnoid hemorrhage throughout the perimesencephalic cistern (arrow) and along the tentorium (double arrows). The subarachnoid blood in the basilar cisterns has refluxed into the 4th (double arrowheads) and 3rd (arrowhead) ventricles. There is marked hydrocephalus . An arteriovenous malformation (please refer to Figure 8), was the etiology of this subarachnoid hemorrhage.

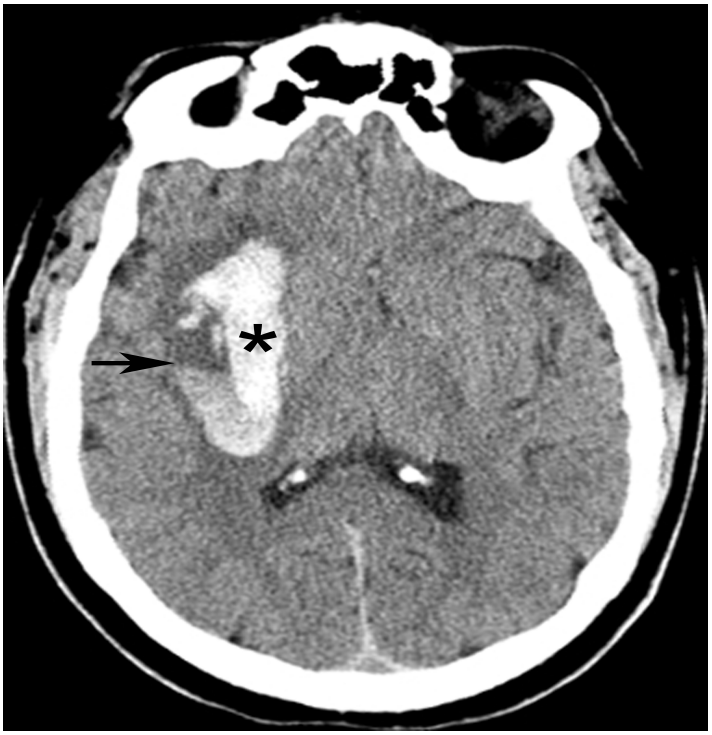

Figure 3. Hypertensive intraparenchymal hematoma. Nonenhanced computed tomography shows a large right basal ganglionic hematoma (*) containing a fluid/fluid level (arrow).

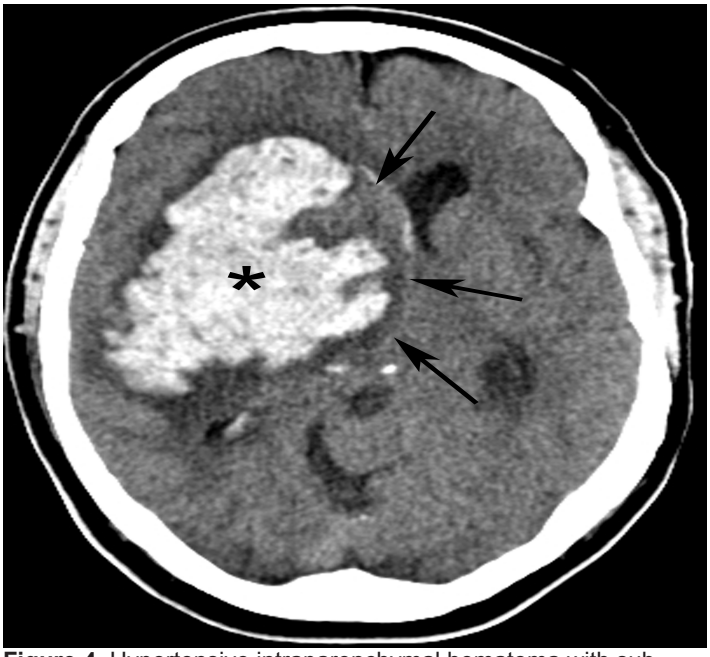

Figure 4. Hypertensive intraparenchymal hematoma with subfalcine herniation. Nonenhanced axial computed tomography demonstrates a large right basal ganglionic hypertensive bleed (*) with mass effect and midline shift or a subfalcine herniation to the left (arrows). The frontal horns are part of the lateral ventricles. The right frontal horn is compressed so severely that it is almost completely obliterated. Dilation of the left frontal horn is due to obstructive hydrocephalus – a consequence of compression of the third ventricle.

COMPUTED TOMOGRAPHY ANGIOGRAM

Newer generation multi-slice CT scanners are becoming more readily available.

CT angiogram (CTA) is a minimally invasive study that requires a time optimized rapid injection of intravenous contrast and thin-section helical CT images are obtained in the arterial phase.

Software allows thin-section axial CT images to be reformatted in any plane enabling a more complete evaluation of vessels. Three-dimensional reformations of contrast-enhanced CT angiograms provide clear images of cerebral blood vessels. Imaging of the entire intra and extra cranial circulation beginning at the aortic arch and continuing through the Circle of Willis can frequently be performed within 60 seconds. (Figure 5).[5-7]

The identification of areas of stenosis or occlusion of vessels allows for rapid and accurate diagnoses and decisions in the clinical arena. CT angiograms that identify occluded blood vessels in the brain assist the neurologist and emergency physician in treatment decisions.

When the CT angiogram reveals an occlusion of a major cerebral blood vessel within the three hour time limit, then the decision to administer intravenous tissue plasminogen activator (TPA, specifically alteplase) can be made with more confidence as the cause of the stroke is more accurately

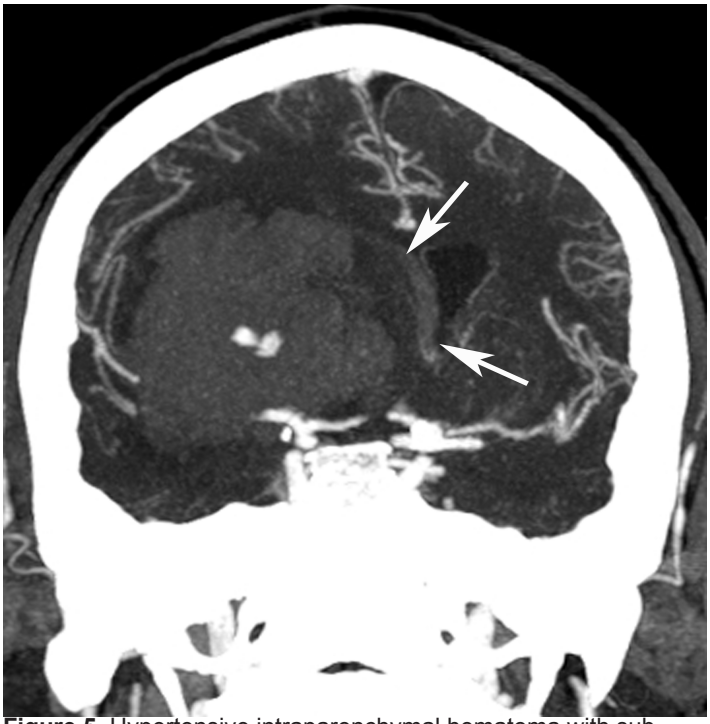

Figure 5. Hypertensive intraparenchymal hematoma with subfalcine herniation. Coronal two-dimensional reconstruction from a computed tomography angiogram demonstrates transfalcine herniation (arrows) to the left due to large right sided intraparenchymal hemorrhage. Same patient as Figure 4.

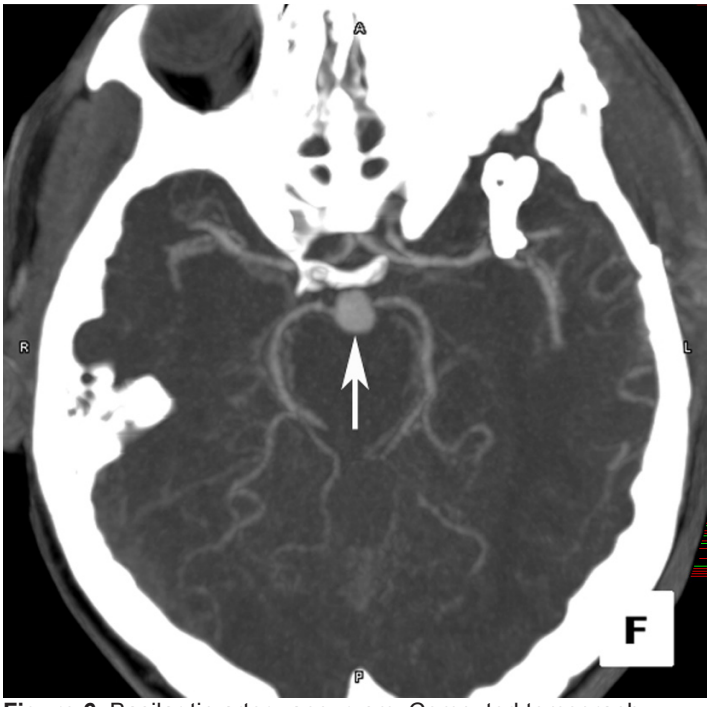

Figure 6. Basilar tip artery aneurysm. Computed tomography angiogram in the axial projection demonstrates a focal basilar tip artery aneurysm (arrow).

identified. Intra-arterial administration of thrombolytics or mechanical clot removal using catheter angiography might be beneficial in patients presenting later than three hours if a large and accessible thrombus is identified and it becomes the decision of the skilled interventional neuroradiologist or neurosurgeon to consider these patients for experimental therapy. The identification of extracranial carotid arterial disease on the CTA and the visualization of the aortic arch might provide the cause of an ischemic event. For example, when a carotid dissection is identified on the CTA, then more aggressive mechanical thrombolytic therapy would not be indicated.[7]

In addition to identifying stenotic and occluded blood vessels the CTA can provide images of aneurysms (Figures 6 and 7) and other vascular abnormalities such as arterio-venous malformation (AVM) and their feeding arteries and veins (Figure 8).[4] Rotation of these images can also assist stroke neurologist, interventional neuroradiologist and surgeons in planning operations and procedures.

Many comprehensive stroke centers are beginning to use this technique in all acute stroke patients. The most important limitations to contrast enhanced CT are prior allergic reactions and patients with underlying renal insufficiency.

COMPUTED TOMOGRAPHY PERFUSION

CT perfusion is more widely available than magnetic resonance imaging (MRI) and can be performed quickly on any standard helical CT scanner right after unenhanced CT. During CT perfusion a rapid intravenous infusion of contrast is administered and sections of the brain are repeatedly imaged. Based on the total amount and speed that blood flows to different vascular territories of the brain this technique can assist in identifying a stroke and potential areas of reversible and salvageable brain tissue in the ischemic penumbra.[7]

The cerebral blood flow (CBF) is equal to the cerebral blood volume (CBV) divided by the mean transit time (MTT). The MTT is the time difference between the arterial inflow and venous outflow.[7-10] MTT is the most sensitive measure used to evaluate for flow abnormalities. It is prolonged in conditions such as hypotension along with occluded and stenotic blood vessels. Time –to –peak (TTP) is sometimes used in place of MTT.

During a stroke, the area of the brain undergoing infarction has both decreased CBF and CBV.[3] Decreased total CBV is the most specific indicator for an area actually undergoing irreversible ischemia or infarct and is non salvageable.

Areas of the brain that are at risk for injury known as the ischemic penumbra show decreased CBF with normal to increased CBV. This potentially salvageable area of the brain must have an intact cerebral autoregulation system to maintain homeostasis. Cerebral autoregulation causes the dilation of the collateral blood vessels and increases the CBV to the areas of the brain that are compromised by decreased CBF

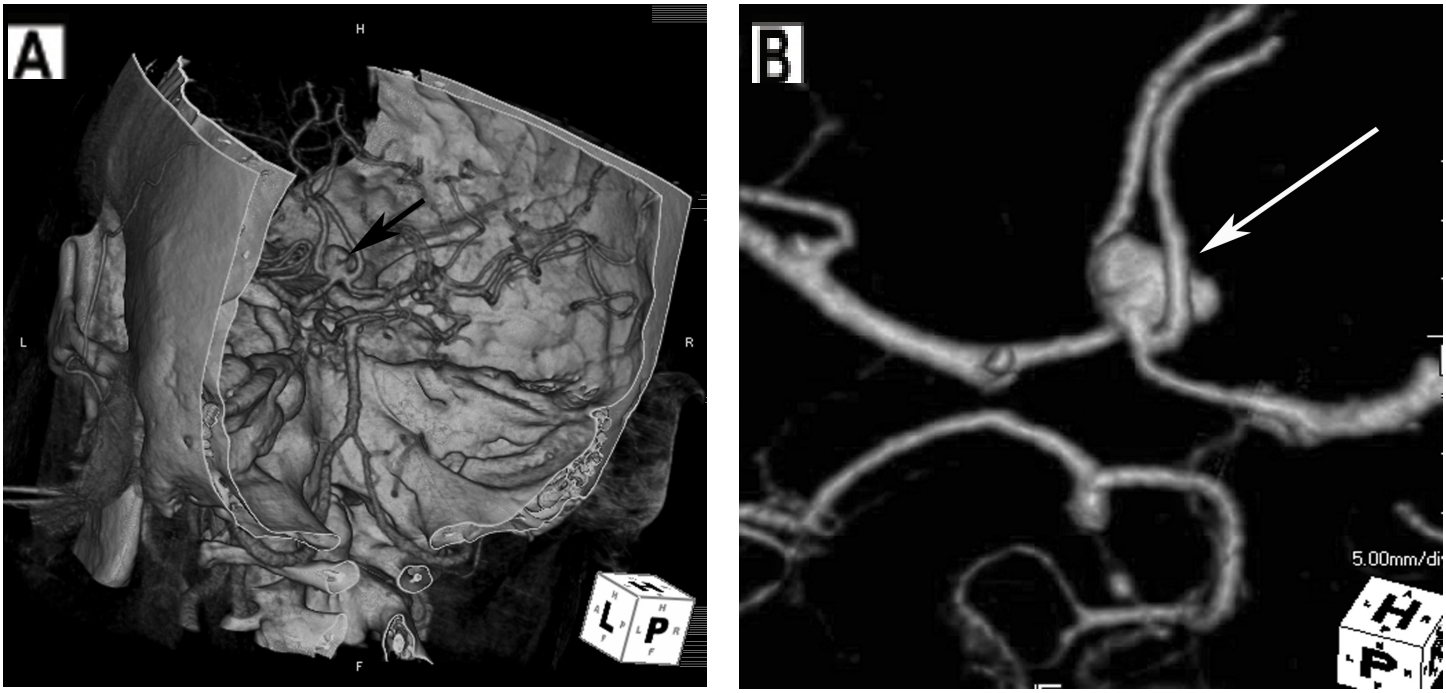

Figure 7. Anterior communicating artery (ACoA) aneurysm. (A) Cerebal computed tomography angiogram (CTA) in three-dimensional projection with skull surface overlay demonstrates an anterior communicating artery aneurysm. (B) Dedicated images from CTA of the Circle of Willis isolates the aneurysm (arrow).

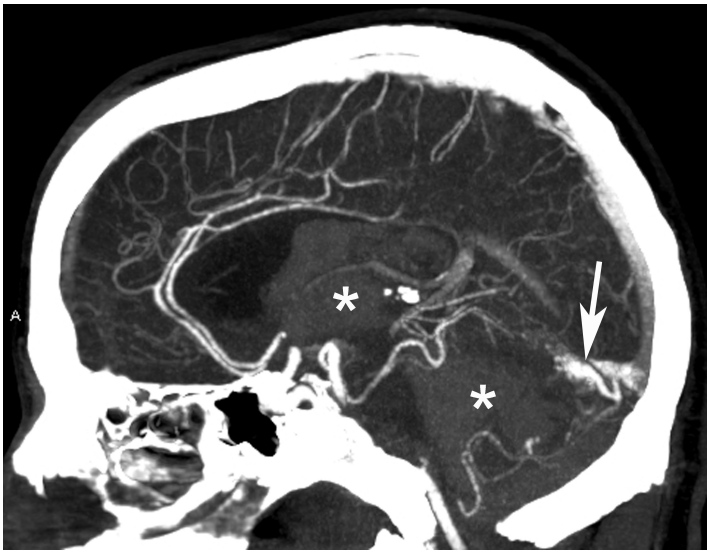

Figure 8. Subarachnoid and intraventricular hemorrhage due to vascular malformation. Sagital reformatted images from computed tomography angiogram demonstrates the enhancing vascular malformation (arrow), which was the etiology of the intraventricular hemorrhage (same patient in Figure 2).

(Figure 9).[3] As an example, tissue distal to the occluded vessel (usually the MCA), receives collateral flow from adjacent non occluded vessels (ACA and PCA). Perfusion to these tissues is characterized by delay in arrival of the contrast bolus but preservation of the total amount of contrast reaching the brain tissue. CT perfusion imaging can identify the penumbra, and it has been used in case reports and studies to guide the treatment of patients in which there is an unknown time of stroke onset, awakening stroke or when the patient cannot communicate the time of onset due to aphasia. These patients may still benefit by intravenous, intra-arterial or mechanical reperfusion. The technique can also be used to evaluate for secondary vasospasm in patients who have sustained subarachnoid hemorrhage.[4]

CT perfusion does have some limitations for it requires multi-detector CT (MDCT) and a special software package which needs to be set up by trained technologist. The software package that is used for CT perfusion analyzes the images obtained and color coded maps representing many levels of the brain are produced to help differentiate the potential cause of the flow abnormalities. Current CT perfusion technology is limited to two slices through the brain. (Higher row MDCT= 64 slice or greater) will be able to image a greater volume of brain tissue. CT perfusion produces a greater amount of radiation exposure to the levels being imaged- roughly 40 CT slices through the same level of the cranium. If the perfusion CT software and equipment is not set up correctly, massive radiation doses may result.

MAGNETIC RESONANCE IMAGING

Conventional brain MRI studies can take up to one hour to complete. The study is not very good at detecting cytotoxic or intracellular edema that is seen in the acute or less than 24 hour phase of stroke. Standard MRI images (T1 and T2)

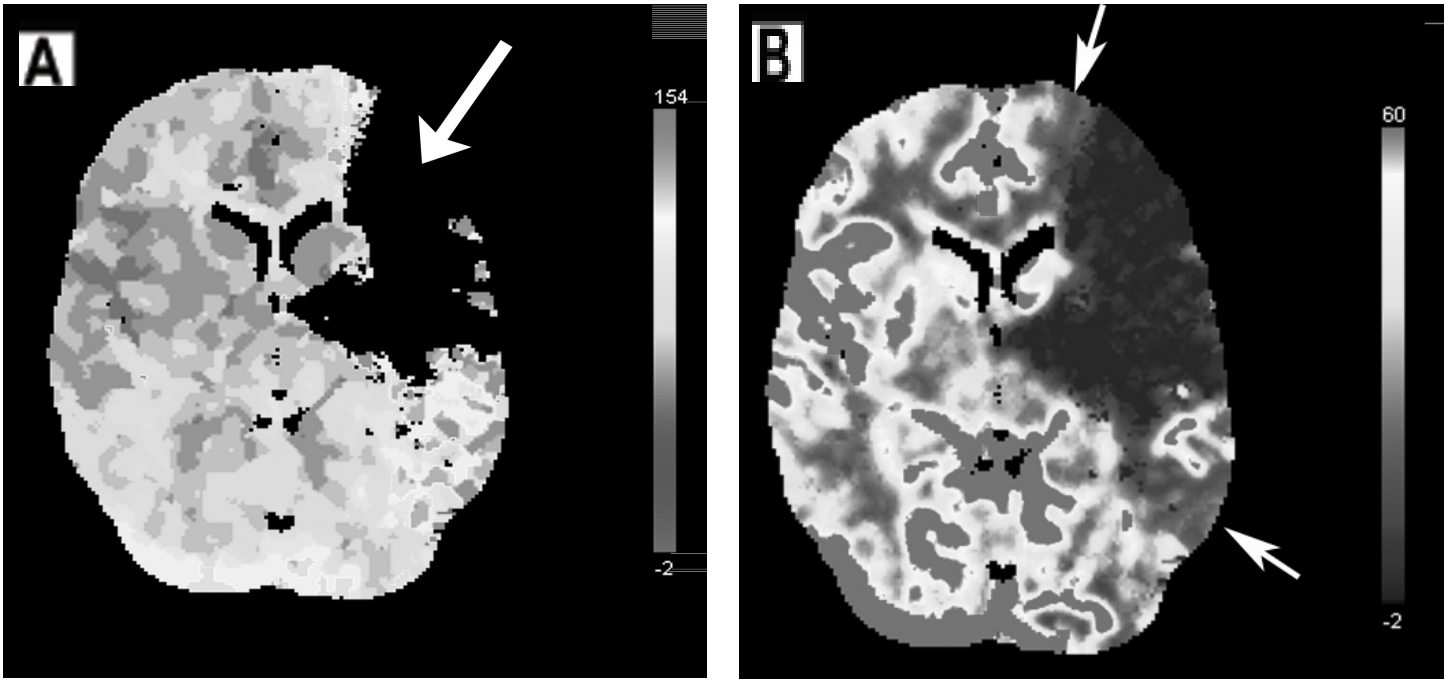

Figure 9. Left middle cerebral artery infarct. (A) Regional cerebral blood flow map from computed tomography perfusion shows a large perfusion defect in the left frontal and temporal lobes, evidenced by a lack of color display. (B) Regional cerebral blood volume map demonstrates a penumbra of decreased perfusion (indicated with arrows around blue areas) surrounding the defect (purple), indicating potentially reversible ischemia about the perfusion defect.

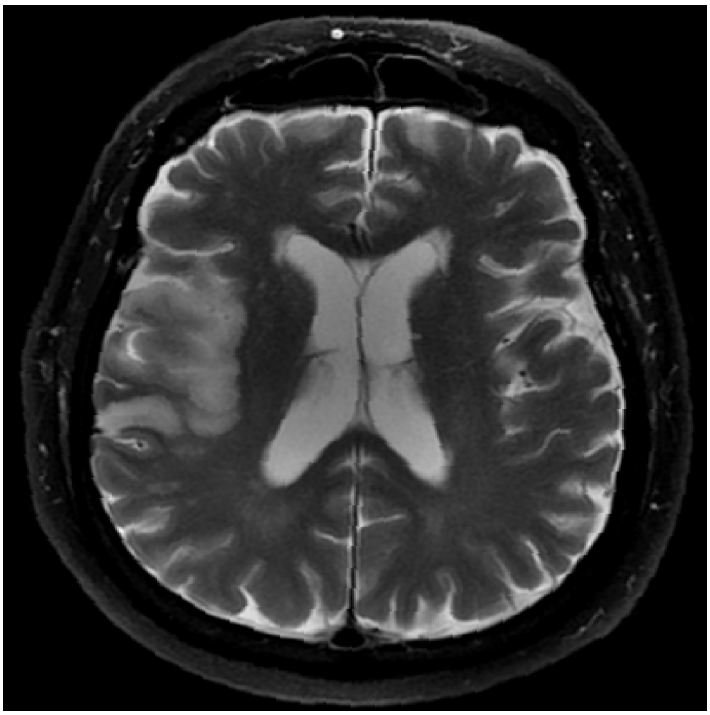

Figure 10. Right middle cerebral artery infarction. Fast spin echo T2-weighted fat suppressed image demonstrates increased signal intensity and effacement of the right temporal lobe, consistent with sub-acute infarct.

are good at detecting vasogenic edema that is present in the subacute phase of stroke and is seen at greater than 24 hours to several days. Fast spin echo T2- weighted sequences can clearly demonstrate areas of edema not visible on the CT and can help identify a subacute stroke as seen in (Figure 10). Fluid attenuated inversion recovery (FLAIR) sequences are designed to suppress signal from the CSF so that it will appear dark. FLAIR images provide good conspicuity of acute subarachnoid hemorrhage, as compared to conventional T-1 and T-2 weighted images and are useful in the initial evaluation of the acute stroke patient suspected of having a subarachnoid hemorrhage. Subarachnoid hemorrhage appears bright on FLAIR images and so becomes readily apparent.

The gradient recalled echo (GRE) sequence is also useful for the detection of blood products. Hypointensity due to paramagnetic effect of the hemosiderin, otherwise known as "blooming," affects the magnetic field and decreases the signal. Therefore blood appears "black" on GRE images (Figure 11).[11]

MAGNETIC RESONANCE DIFFUSION

MR diffusion is a technique that is having a dramatic affect on the approach and management of acute ischemic stroke patients.[12-14] MR diffusion is diffusion weighted images (DWI) and can be obtained within 10 minutes at some centers and dramatically alter care, so the clinical determination of ischemic stroke can be confirmed quickly. DWI is used to detect early ischemic changes (acute stroke; early ischemic change; cytotoxic edema) with greater conspicuity than standard MRI. MRI with diffusion is quickly becoming the gold standard in acute stroke imaging. Once a hemorrhagic stroke has been excluded by CT, MR diffusion improves stroke detection from 50% to more than 95%.[3-4,14] Diffusion

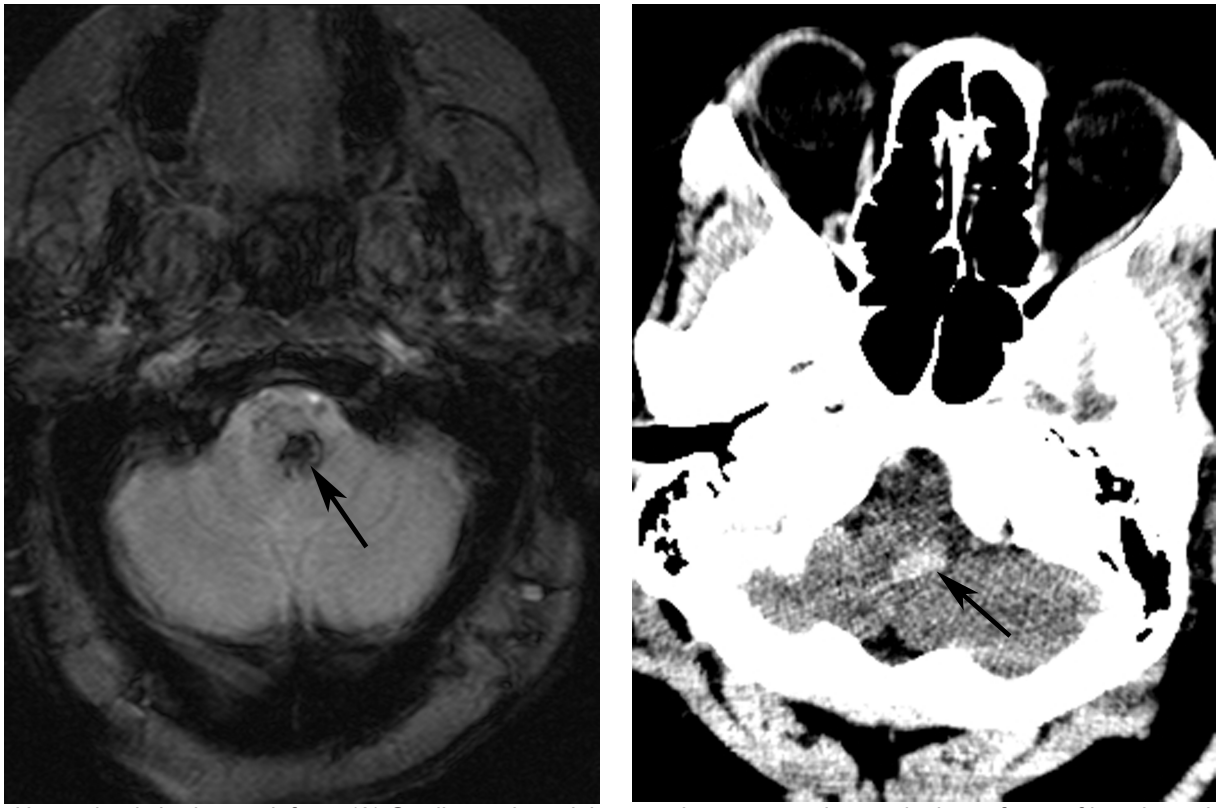

Figure 11. Hemorrhagic brainstem infarct. (A) Gradient echo axial magnetic resonance image depicts a focus of hypointensity due to paramagnetic effect of the hemosiderin, otherwise known as "blooming." (B) Axial computed tomography through the brainstem demonstrates a corresponding hyperattenuating focus of hemorrhage (arrow) in the brainstem.

MR noninvasively detects ischemic changes within minutes of stroke onset.

It is the ability to quantify the motion of water molecules that makes the process of diffusion weighted MRI possible. Normal uninjured neuronal cells allow for the equal movement of water between healthy cells. During an acute ischemic stroke cytotoxic intracellular injury to the neuronal cell occurs and intracellular water accumulates in the injured cells as the cells ability to allow for the water to diffuse out is decreased. The difference in the water content and the diffusion of water between injured and uninjured cells can be measured and allows for the identification of ischemic cells on diffusion MRI images. These areas of damage appear bright on MR diffusion images. [7,14]

In order to help identify areas of ischemia, apparent diffusion coefficient (ADC) maps are used and areas that are bright on diffusion and dark on ADC are consistent with acute infarct (Figure 12). It is necessary to use ADC maps because some areas of high signal such as vasogenic edema can appear bright on the initial diffusion image signaling. This is because the diffusion sequence is T-2 based and this "shine through" can cause some bright signals, but these areas of high signal that are not secondary to acute infarct are easily identified on the ADC maps and allow for an accurate initial diagnosis. Over time, the appearance of the diffusion and ADC abnormalities will reverse as the stroke moves into a subacute phase (more than 24 hours to 5 days).[4,7] The specific injury patterns that are identified on MRI diffusion help the skilled neuroradiologist to date the time of onset, progression and resolution of strokes.

MR diffusion imaging allows clinicians who are evaluating challenging patients with neurological deficits to differentiate between those who actually have an acute ischemic event versus those who do not. It can therefore help differentiate a stroke from other stroke mimics such as a hemiplegic migraine headache, Todd's paralysis (seizure) and peripheral or cranial nerve disorders.

MAGNETIC RESONANCE IMAGING PERFUSION

Pefusion imaging of the entire brain is one of the main advantages of perfusion-MR (perfusion weighted imaging=PWI), in which MMT or TTP perfusion maps are generated for the entire brain. [3] Like CT perfusion, it can identify the ischemic penumbra. The ischemic penumbra is the difference between the DWI defect (cytotoxic edema-irreversible ischemia- the ischemic core) and the perfusion defect-analogous to MTT or TTP). The penumbra is the DWI-PWI mismatch. The accurate identification of this ischemic penumbra will help guide future ischemic stroke therapy and potentially aide in extending the time window for treatment.

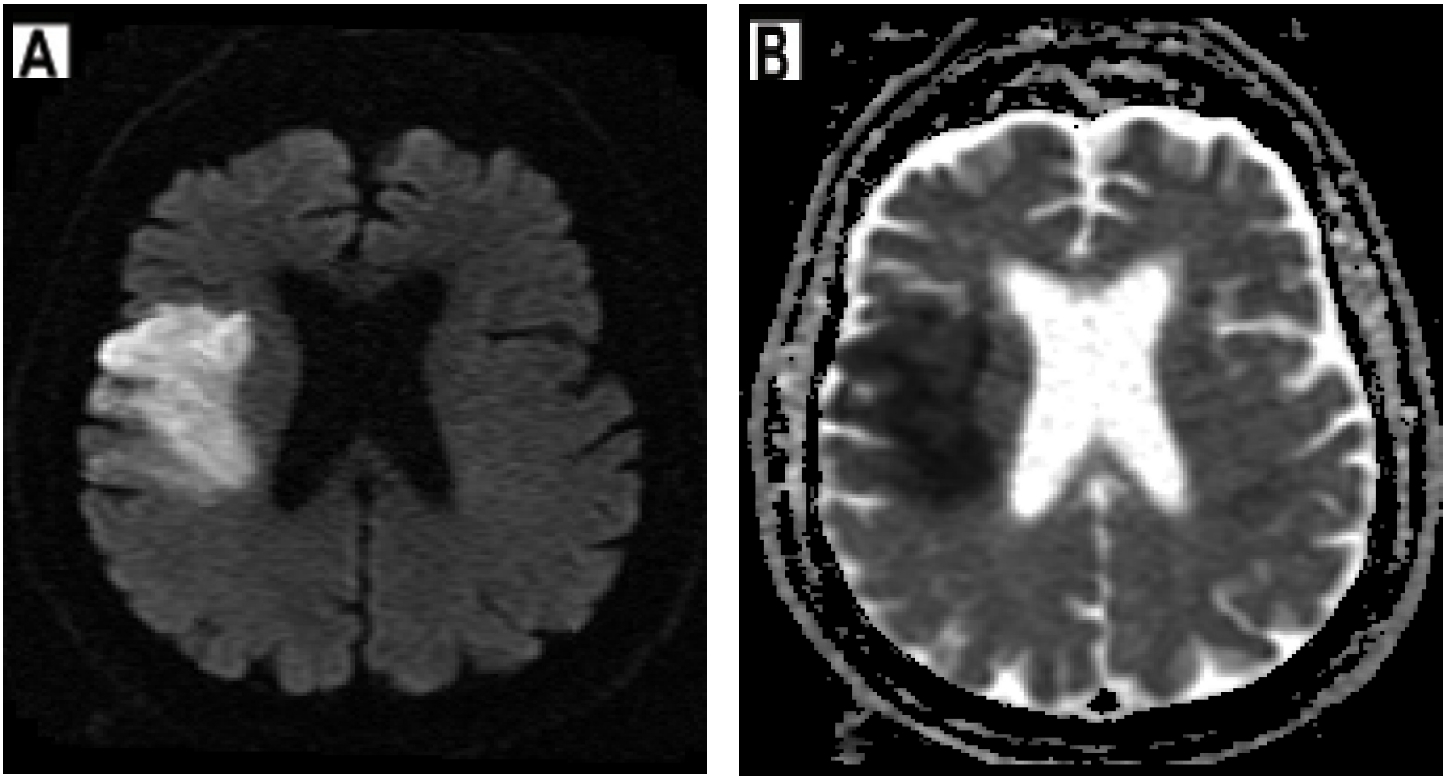

Figure 12. Right middle cerebral artery infarction. (A and B) Restricted water diffusion in the region of infarct results in an increased signal intensity on diffusion-weighted imaging (A) and decreased signal on apparent diffusion coefficient imaging (B).

MAGNETIC RESONANCE ANGIOGRAPHY

Magnetic resonance angiography (MRA) can be performed in combination with brain MRI in the setting of stroke to help guide therapeutic decision making. It can detect high grade atherosclerotic lesions in the neck and head. It is also helpful for detecting less common causes of ischemic stroke such as carotid and vertebral artery dissection, fibromuscular dysplasia, and venous thrombosis. As with other forms of MRI imaging it cannot be used in patients with pacemakers, some metallic implants, allergy to MR contrast agents, and in those with severe claustrophobia.

SONOGRAPHY

Transcranial doppler (TCD) examination of the head and doppler imaging of the carotid vessels are helpful adjunct modalities in the evaluation of acute stroke.[15-17] This may be especially useful for patients who are unstable. TCD can be performed for the evaluation of the basal intracranial arteries in patients with acute ischemic stroke who are not eligible for standard imaging techniques, such as CTA or MRA (Figure 13). The arteries best evaluated are those at the base of the brain (MCA, anterior cerebral artery, carotid siphon, vertebral artery, basilar and ophthalmic artery). However, up to 30% of patients are unable to be examined by this technique due to impedance from the temporal bone.[15] The primary application of TCD are to detect and quantify intracranial vessel stenosis, occlusion, collateral flow, embolic events, and cerebral vasospasm (particularly after SAH). Carotid Doppler imaging has been proven to be accurate in the assessment of degree of occlusion of the extracranial portions of internal carotid artery.

ANGIOGRAPHY

Catheter- based cerebral angiography or digital subtraction angiography (DSA) is the standard against which all non-invasive assessments of carotid luminal narrowing are commonly compared and remains the gold standard for the detection of many types of cerebrovascular disease. Angiography is a dynamic study and is a useful tool to visualize slow blood flow through a constricted vessel and delayed filling of capillary vessels.[18] In addition, cerebral angiography can identify and allow the specialist to treat occluded or narrowed vascular segments and vascular malformations (Figures 16 and 17).

CONCLUSION

An accurate and practical understanding of imaging modalities is essential for the management of the acute stroke patient. Due to its wide spread availability and speed, unenhanced CT scan remains the initial study of choice for evaluating an acute stroke patient - it is used for inclusion criteria and to rule out hemorrhage. After bedside point of care testing is performed to establish the baseline creatinine, CT angiography can be obtained to give added information on stenotic and occluded blood vessels. If the CTA demonstrates

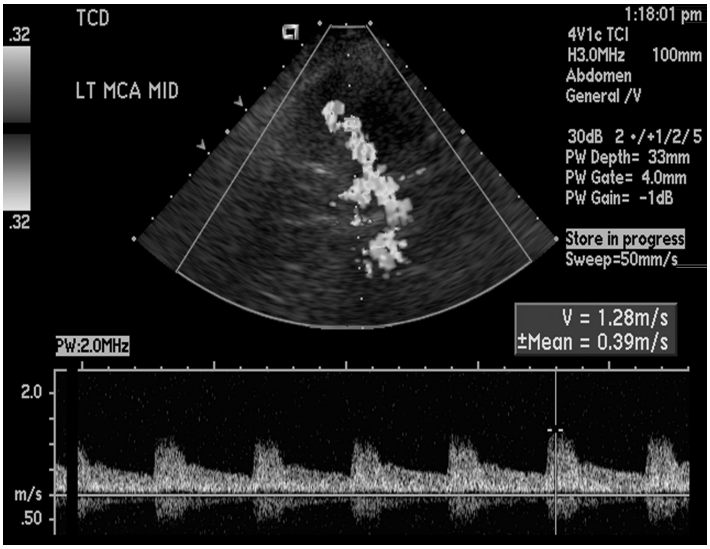

Figure 13. Normal transcranial Doppler. Color Doppler imaging of the left middle cerebral artery demonstrates normal flow and waveforms, with a peak velocity of 1.28 m/s.

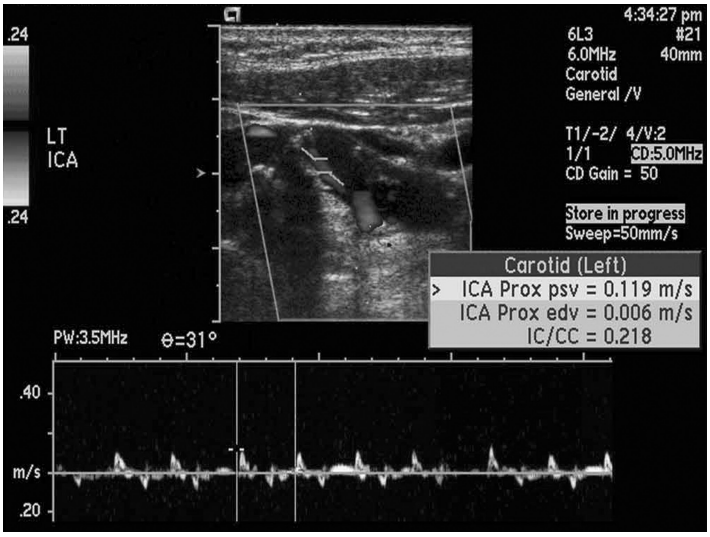

Figure 14. Acute high-grade internal carotid artery (ICA) occlusion. Longitudinal color Doppler image through the internal carotid artery demonstrates a "string" sign, with very minimal flow through the ICA.

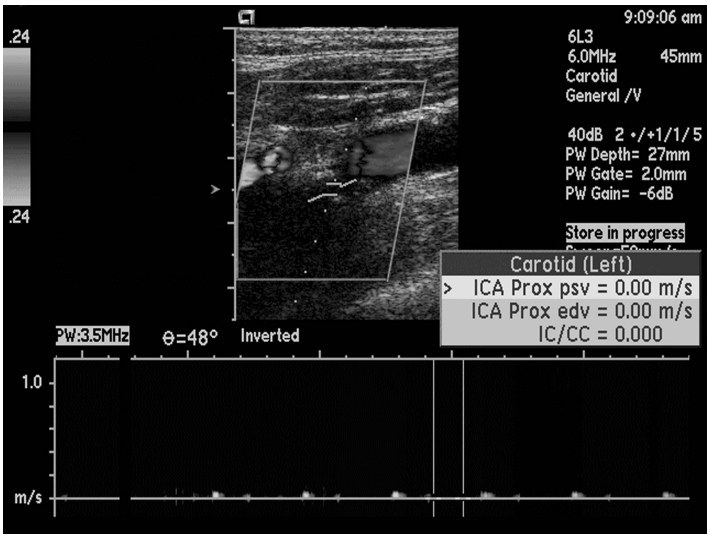

Figure 15. Acute complete internal carotid artery occlusion. Longitudinal sonogram of the left internal carotid artery demonstrates complete flow void, with no perceptible waveforms.

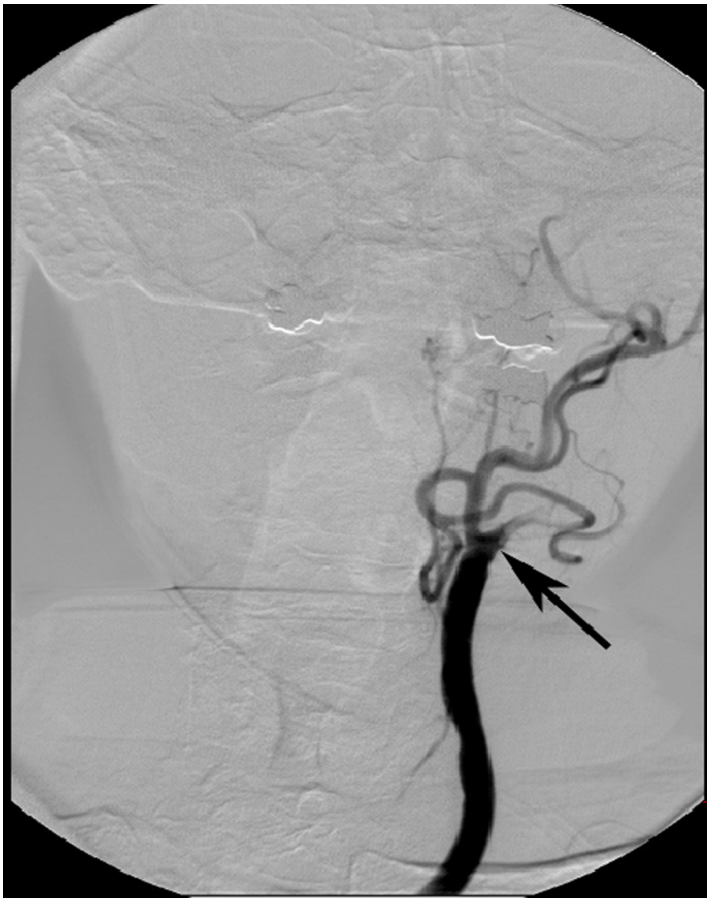

Figure 16. Internal carotid artery occlusion. Frontal projection from left cerebral angiogram delineates complete occlusion of the left internal carotid artery to the level of the common carotid artery (arrow). Note the normal opacification of the external carotid artery branches.

a large or significant thrombus burden *outside the three hour time window* then intrarterial or mechanical thrombolysis may be an indicated treatment which may be performed by the skilled interventional neuroradiologist.

CT and MRI perfusion imaging can assist in identifying a stroke and potential areas of reversible and salvageable brain tissue in the ischemic penumbra.[7] Identifying patients initially *outside of the three hour window* or in those which the time of onset is uncertain who have an area of potentially reversible ischemia may still benefit by intravenous, intra-arterial or mechanical reperfusion.

Traditional MRI remains a vital tool in the evaluation of the subacute stroke patient, due to its excellent soft tissue

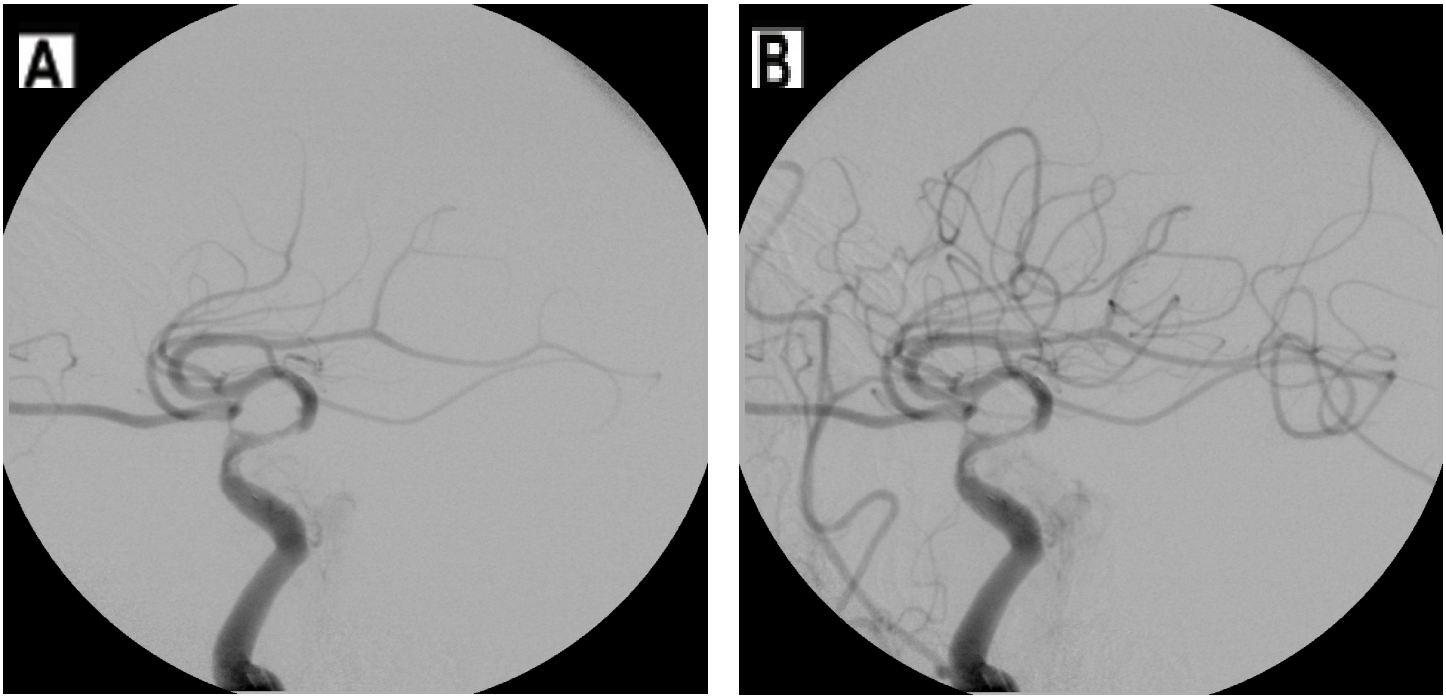

Figure 17. Left middle cerebral artery (MCA) recanalization. (A) Initial lateral image from cerebral angiogram demonstrates a paucity of vessels in the MCA distribution. (B) Repeat lateral angiogram after intra-arterial lysis demonstrates recanalization of flow and normalized perfusion.

contrast. Specialized MRI techniques are also essential to exclude hemorrhage in patient's whom SAH is suspected. MR with diffusion imaging can non-invasively detect ischemic changes and can be obtained within 10 minutes at some centers and dramatically alter care, so the clinical determination of ischemic stroke can be confirmed quickly. MRI with diffusion is quickly becoming the gold standard in acute stroke imaging but its use is limited because of the universal lack of access to this expensive equipment and technology, along with capable and experienced neuroradiologist to interpret the results.

MRA is helpful for detecting less common cause of ischemic stroke such as carotid and vertebral artery dissection, fibromuscular dysplasia, and venous thrombosis. It also aides in the detection of underlying aneurysms.

Sonography is currently an adjunct modality for stroke evaluation in certain settings. Angiography remains the gold standard against which all non-invasive assessments of carotid luminal narrowing and many types of cerebrovascular diseases are identified and treated . Catheter angiography is an important tool in the hands of skilled interventionalist and continues to be the modality to which new therapies in stroke care are constantly evolving and being compared.

While the ACR prefers MRI to CT for acute stroke it is not currently available and practical for most centers. The recommended MRI sequences are T1,T2, FLAIR, GRE (for Blood), DWI for acute ischemia, MRA, and PWI (for penumbra imaging). Most United States emergency departments do not have MR available for acute stroke .It is not uncommon in the ED to use DWI- MRI when the diagnosis of an acute stroke is unclear and it can help confirm the diagnosis. [3]

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Excited Delirium

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Excited (or agitated) delirium is characterized by agitation, aggression, acute distress and sudden death, often in the pre-hospital care setting. It is typically associated with the use of drugs that alter dopamine processing, hyperthermia, and, most notably, sometimes with death of the affected person in the custody of law enforcement. Subjects typically die from cardiopulmonary arrest, although the cause is debated. Unfortunately an adequate treatment plan has yet to be established, in part due to the fact that most patients die before hospital arrival. While there is still much to be discovered about the pathophysiology and treatment, it is hoped that this extensive review will provide both police and medical personnel with the information necessary to recognize and respond appropriately to excited delirium. [West J Emerg Med. 2011;12(1):77-83.]

INTRODUCTION

Excited delirium (EXD), first described in the mid 1800's, has been referred to by many other names – Bell's mania, lethal catatonia, acute exhaustive mania and agitated delirium.[1] Regardless of the label used, all accounts describe almost the exact same sequence of events: delirium with agitation (fear, panic, shouting, violence and hyperactivity), sudden cessation of struggle, respiratory arrest and death.[2] In the majority of cases unexpected strength and signs of hyperthermia are described as well.[3,4].While the incidence of EXD is not known, the purpose of this review is to identify what is known or suspected about the pathophysiology, outcomes and management options associated with EXD to assist medical professionals in the future.

Issues Regarding EXD

EXD has gained increasing public attention recently due to the number of post-mortem explanations offered by medical examiners regarding the death of individuals being restrained by police or being taken into custody. This diagnosis has caused concern because EXD is not a currently recognized medical or psychiatric diagnosis according to either the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IVTR) of the American Psychiatric Association or the International Classification of Diseases (ICD-9) of the World Health Organization. Likewise, the authors of one review article found enough evidence in the literature to suggest that excited delirium, rhabdomyolysis and neuroleptic malignant syndrome might represent the clinical spectrum of a single disease.[5] Although more research is needed to elucidate cause and effect, it is important to note that a lack of recognition of the condition in the context of law enforcement activities does not negate the significance of the behavioral and physical signs referred to as EXD. For instance, one important study found that only 18 of 214 individuals identified as having EXD died while being restrained or taken into custody.[6] If anything, the possible association with other life-threatening syndromes only gives impetus to the need for critical emergency medical intervention when encountering a person thought to be in a state of excited delirium.

Background

Although reports of patients with similar symptoms first appeared in the 19th century, the first modern mention of EXD was in 1985.[3] The presentation of excited delirium occurs with a sudden onset, with symptoms of bizarre and/or aggressive behavior, shouting, paranoia, panic, violence toward others, unexpected physical strength and hyperthermia. An extensive review of reported case series reveals that in a majority of cases EXD was precipitated by stimulant drug use and in much fewer cases psychiatric illness (such as mania, depression, or schizophrenia) or systemic illness.[7-9]

Methamphetamine, PCP and LSD have been reported in a few series, but by far the most prevalent drug of abuse found on toxicology screening was cocaine.[10] Since the victims frequently die while being restrained or in the custody of law enforcement, there has been speculation over the years of police brutality being the underlying cause. However, it is important to note that the vast majority of deaths occur suddenly prior to capture, in the emergency department (ED), or unwitnessed at home.

Prior to 1985 most reported cases of sudden death from cocaine intoxication involved "body stuffers" who died secondary to massively high exposure to the drug after packets they were carrying burst. A report published by Wetli and Fishbain [3] in 1985 was one of the first case series to examine recreational cocaine users who died following episodes of excited delirium. They noticed that these deaths differed in both presentation and average blood cocaine concentrations from typical cocaine overdose fatalities. In fact, cases of agitated delirium were often associated with lower blood levels of cocaine. Explorations by Pollanen et al[9] and Ruttenber et al[11] showed blood levels of cocaine in EXD cases to be similar to levels found in recreational cocaine users and much lower than levels found in people who died from cocaine associated intoxication. Moreover, the reports found that the blood levels of benzoylecgonine, the primary metabolite of cocaine, in the cocaine-associated EXD cases were higher than in recreational users, suggesting the cocaine use prior to death was consistent with recent "binge" use. More recently, Stephens et al.,[12] in an analysis of the significance of cocaine upon a specific death, confirmed that a pattern of chronic cocaine use characterized by repeated binges is associated with the development of fatal EXD.

PATHOPHYSIOLOGY

Cocaine has many neurotransmitter affects on the brain, including the blockade of all monamine neurotransmitters via its interaction with the various transporters. The reinforcing or addictive properties of cocaine are primarily attributed to increased dopamine levels. Dopamine is an essential neurotransmitter in several neural pathways regulating movement, hypothalamic function, positive behavioral reinforcement and higher cognitive function. The mesolimbic pathway, which connects the nucleus accumbens and tegmentum, is most critical for reinforcement and addiction to psycho-stimulants. Several researchers have suggested that cocaine use may cause aberrant dopamine processing in the mesolimbic pathway and elsewhere in the brain, resulting in hyperactivity and hyperthermia.[11,13] Additionally, in cases of EXD, dopamine processing has been shown to be further altered compared to non-psychotic cocaine users. Recent research has identified several possible explanations for this critical difference.

First, Mash et al[14] discovered evidence of increased alpha α-synuclein, a native protein and major component of Lewy bodies in Parkinson's disease, in midbrain dopamine neurons of chronic non-psychotic cocaine users.[2] *In vivo*, increased binding of α-synuclein to dopamine transporters has been shown to increase dopamine uptake and dopamine mediated apoptosis, leading to irreversible neuroadaptive changes. The authors suggested that the increased α-synuclein deposition might occur as a protective response due to high dopamine recycling and oxidative stress from cocaine abuse. Their postmortem research demonstrated that chronic non-psychotic cocaine users, compared to control non-drug users, had markedly elevated presynaptic α-synuclein levels in the substantia nigra and ventral tegmental area of the midbrain. However, compared to the same control group, victims of fatal EXD showed decreased levels of α-synuclein in the substantia nigra and only slightly increased levels in the ventral tegmental area. This discovery suggests that EXD victims might have a different pattern of α-synuclein regulation and perhaps lack normal compensatory measures for dealing with rapidly elevating dopamine levels.

Second, multiple studies have documented dopamine transporter binding sites that are increased in human chronic cocaine users.[15-17] Mash et al[18] used *in vitro* autoradiography and ligand binding studies to map and measure D3 dopamine and kappa 2 opioid receptors in brain tissue from postmortem cocaine overdose victims and compared them to fatal EXD victims. The D3 receptor subtype has a distinctive neuroanatomy pattern in the normal human brain, with high densities in areas associated with the pattern-building, euphoric effects of cocaine, such as, the nucleus accumbens and limbic sectors of caudate and putamen. The authors found that chronic cocaine abuse lead to an adaptive elevation of D3 dopamine receptors (2-3 times vs. control) and kappa 2 opioid receptors (2 times vs. control) in the nucleus accumbens and associated limbic regions. By contrast, cocaine abusing EXD victims did not demonstrate increased density for D3 receptor binding. This finding mirrored similar results by Staley et al[19] that failed to demonstrate an elevation in dopamine transporters in the striatum of EXD victims versus aged-matched drug-free control patients. Mash et al[18] suggested that the lack of compensatory changes in the EXD victims could be related to concurrent psychiatric co -morbidity, recent "binge" cocaine use, or aberrant molecular processing of D3 receptor mRNA. Interestingly, different mRNA species have been found in the cerebral cortices of chronic schizophrenia patients, which raise the possibility that similar alterations in D3 receptor processing could be involved in EXD victims.[20]

Third, further investigation by Mash et al[22] focused on the functional activity of dopamine transporters, which were previously found to be elevated in the limbic system of chronic cocaine abusers.[18,21] Using cryopreserved tissue samples from aged-matched chronic cocaine users (n=10), EXD victims (n=8) and control subjects (n=10), the authors quantified the number of dopamine transporters in parallel to dopamine uptake and discovered dopamine uptake was

twofold in the ventral striatum from chronic cocaine users versus aged-matched controls. Victims of fatal EXD failed to demonstrate an increase in dopamine transport function, despite having a history of cocaine use and post-mortem blood elevations of cocaine and benzoylecgonine. Based on the results, chronic cocaine use seems to cause a compensatory increase in dopamine transporters, which would decrease the amount of dopamine available to potentially over stimulate the post-synaptic receptors. This effect, which may be neuron protective, is lacking in fatal EXD victims.

In addition to altering dopamine reuptake directly, cocaine has been shown to potently inhibit serotonin reuptake, thus elevating synaptic levels of the neurotransmitter.[22] Despite the relative certainty that dopamine is the primary substrate mediating the reinforcing and addictive properties of cocaine, a study by Rocha et al[23] of dopamine transporter knockout mice suggests the involvement of serotonergic brain regions in the initiation and maintenance of cocaine self-administration and withdrawal symptoms. Meanwhile, serotonin has been implicated as an independent modulator of dopaminergic neurotransmission. Mash et al[24] compared serotonin transporter density in brain tissue from cocaine overdose victims and cocaine-associated EXD victims, finding that the transporters localized to the dopamine rich substantia nigra and striatum in response to chronic cocaine use. Once again, EXD victims failed to display an up regulation of serotonin transporters within aforementioned brain regions.

Lastly, in a 2009 case series of an unprecedented ninety fatal EXD victims, Mash et al[25] conducted a post-mortem quantitative analysis of dopamine transporters and heat shock protein 70. Incident circumstances, force measures, autopsy and toxicology results were determined and controlled in the analysis. Mean core body temperature among the ninety victims was 40.7°Celsius and, although the majority tested positive for cocaine, four had no licit or illicit drugs or alcohol found at autopsy. The authors discovered heat shock proteins were elevated 1.8-4 fold in postmortem brain tissue, confirming that hyperthermia is an associated symptom and indication of fatal autonomic dysfunction in the victims. In addition, dopamine transporter levels were decreased compared to age-matched controls, which correlate with the findings by previous authors of aberrant dopamine signaling in EXD.

These observations demonstrate that cocaine affects a number of different neurochemical substrates in the brain and suggest that chronic exposure may lead to complex neuroadaptations within discrete brain loci. Furthermore, compared to non-psychotic cocaine overdose victims, fatal EXD victims have been shown to possess alterations in neuroanatomy and neurophysiology that may represent a subtype of patient with an altogether unusual genotype and/or phenotype; one characterized by high dopamine levels and a hyperactive autonomic nervous system. This understanding may lead to changes in the recognition, handling and acute treatment of EXD by first responders and emergency physicians.

OUTCOMES

Approximately two thirds of EXD victims die at the scene or during transport by paramedics or police.[26] Victims who do not immediately come to police attention are often found dead in the bathroom surrounded by wet towels and/or clothing and empty ice trays, apparently succumbing during failed attempts to rapidly cool down.[2] It appears that in all cases, victims died of either respiratory arrest or fatal cardiac dysrrhythmia. Diagnoses were supported by postmortem exams showing pulmonary and cerebral edema with nonlethal self-inflicted injuries.[3,10,27,28] The few who live long enough to be hospitalized often succumb to disseminated intravascular coagulation, rhabdomyolysis and renal failure.[2] These fatal cardiopulmonary changes are thought to be the result of increased catecholamine stress on the heart, myocardial hypertrophy, microangiopathy and fatal arrhythmias.[3,10] The proposed cause of these changes is debated.

Since the victims sometimes die in police custody, the most widely publicized proposed causes of death in EXD are taser use and positional asphyxia. No study thus far has been able to demonstrate a causal relationship between Taser use and subsequent individuals' deaths.[10,28] In one study of 32 healthy police volunteers, a 12-lead electrocardiogram was performed at baseline and then repeated within 60 seconds post-exposure to a one to five second shock by the Taser X26.[29] The authors reported no instances of dysrhythmia nor ectopy among the subjects. Furthermore, no statistically significant changes were noted in the QRS duration, QT and QTc intervals. These results corroborate with previous reports using single-lead monitoring to assess cardiac changes before, during, and after Taser activation.[30,31]

As mentioned before, people experiencing EXD are highly agitated, violent, and show signs of unexpected strength so it is not surprising that most require physical restraint. The prone maximal restraint position (PMRP, also known as "hobble" or "hogtie"), where the person's ankles and wrists are bound together behind their back, has been used extensively by field personnel. In far fewer cases, persons have been tied to a hospital gurney or manually held prone with knee pressure on the back or neck.[6,9,10,26] Supporters of the positional asphyxia hypothesis postulate that an anoxic death results from the combination of increased oxygen demand with a failure to maintain a patent airway and/or inhibition of chest wall and diaphragmatic movement.[9,10] This explanation has been further supported by coroners' reports of "positional asphyxia" as the cause of death in multiple fatal EXD cases.

The positional asphyxia theory has been refuted by a series of articles by Chan et al[32] exploring the effect of PRMP on ventilatory capacity and arterial blood gases. In one study of fifteen healthy male volunteers, the authors found a small, but statistically significant decline in forced vital capacity

(FVC), forced expiratory volume in one second (FEV1) and maximal minute ventilation (MVV) comparing sitting to restrained positions. However, there was no evidence of hypoxia (mean oxygen tension [PO2] less than 95 mmHg or co-oximetry less than 96%) in either position, nor was there a significant difference in PCO2, heart rate recovery or oxygen saturation. In another study, the authors sought to determine the effect of adding 25 and 50 pounds weight force on respiratory function of healthy volunteers in the PRMP.[33] Validating earlier results, they found FVC/FEV1 was significantly lower in restrained positions versus sitting, but not significantly different between restrained positions with and without weight force. Furthermore, they found mean oxygen saturation levels were above 95% and mean end-tidal CO_2 levels were below 45 mmHg for all positions, regardless of weight force. Based on these findings, PMRP may result in a transient pattern of restricted pulmonary function, but the lack of evidence for hypoxia or hypoventilation suggests that factors other than body positioning appear to be more important determinants for sudden, unexpected death. Nonetheless, respiratory muscle fatigue resulting from exertion and struggle against restraints (exertion vs. position asphyxia) cannot be excluded nor can potentially fatal pre-existing problems with central cardiac output, oxygen saturation, or oxygen use.[6,26,28,34]

Another potential cause of death is cardio toxicity due to chronic cocaine abuse. Preexisting coronary artery disease appears to account for many of the deaths, as does the contribution of cocaine acting as a potent adrenergic agonist, but the mechanism is likely more complex.[27] A larger case series published in 2006 noted that more than half of EXD fatalities were found to have some degree of cardiovascular disease.[28] Since the majority of deaths occur after prolonged drug use, it is thought that cocaine initiates a series of detrimental changes to the heart that might take years to express. These changes may be due to long-term catecholamine toxicity and include cardiac hypertrophy, microangiopathy and myocardial fibrosis.[35] Electrocardiographic and autopsy studies confirm that the heart weight of cocaine users, is, on average, ten percent greater than expected values.[36,37] It is not clear how cocaine initiates the process of hypertrophy, but it could be due to the direct oxidant effect of cocaine or cocaine-induced hypertension. Either way, research using rats injected with cocaine demonstrated increases in levels of mRNA coding for atrial naturetic factor, collagen and alpha/beta myosin.[38]

Small intramyocardial arteries are often thickened in cocaine users. It is hypothesized that cocaine-induced apoptosis damages the muscular layer of the small vessels or that the damage is once again due to the direct oxidant effect of cocaine.[39] The smaller artery lumen may lead to a mismatch in blood flow supply-demand, and ultimately under perfusion whereby the myocardium is not receiving enough blood and becomes ischemic. Lastly, cocaine users' hearts often resemble those of patients with heart failure secondary to pheochromocytoma. The high levels of catecholamine, particularly norepinepherine, seem to induce a diffuse pattern of discrete fibrotic lesions, which tend to favor reentry and the induction of arrhythmias.[40]

Hypertrophied hearts with diffuse fibrosis and microangiopathy utilize oxygen less efficiently and are more likely to have disordered electrical conduction. During times of increased stress, the myocardium becomes ischemic and should have a lower threshold for fibrillation. Unfortunately, few case series have been able to publish the initial cardiac rhythms found at the scene of cocaine-associated EXD fatalities. One report documents 18 fatal EXD cases with 13 primary cardiac rhythms confirmed by emergency personnel.[6] Only one victim was confirmed to have ventricular tachycardia and none were found to have ventricular fibrillation. If primary arrest was strongly associated with sudden death in excited delirium, it would be expected that more victims would have presented with the above-mentioned abnormal rhythms. However, this case series was limited because the exact time delays in determining initial cardiac rhythms on arrival of emergency medical services to the scene was not available; thus, it is impossible to calculate how many of the patients might have progressed from ventricular tachycardia/fibrillation to asystole. Nevertheless, the molecular, cellular and anatomic alterations induced by chronic higher-dose cocaine use might explain why very low cocaine levels can be lethal in EXD victims.

MANAGEMENT

While our understanding of EXD is expanding, the disorder still presents significant challenges to emergency first responders and physicians. Recent research has demonstrated unique cellular and neurochemical alterations in EXD victims, leading to dopamine excess and autonomic hyperactivity. EXD victims display extreme agitation, aggression, unexpected physical strength and florid psychosis. Emergency physicians must recognize the danger posed by these patients and should act in an expeditious and aggressive manner to avoid medical complications including metabolic acidosis, rhabdomyolysis, hyperthermia, multisystem failure and/or death. To address these clinical findings, we propose a treatment protocol that includes rapid sedation, followed closely by external cooling, intravenous (IV) fluids, monitoring, and treatment of potential medical complications.

Given the violent and unpredictable nature of EXD victims, rapid sedation is likely essential to positive outcomes. Furthermore, if autonomic hyperactivity and aberrant dopamine processing is to blame for the clinical presentation of EXD, then the ideal drug(s) needs to "turn off" the catecholamine cascade and rapidly sedate the patient. Several types of drugs could fulfill these requirements. Neuroleptics, benzodiazepines, or both in combination are commonly used in the management of agitated patients; however, to date, there

are no published double-blind, randomized, placebo-controlled trials to confirm the efficacy and safety of antipsychotic medications to manage acute delirium.[41] One study of 111 violent and agitated patients by Nobay et al[42] compared efficacy and side effect profiles of intramuscular (IM) midazolam (5mg), lorazepam (2mg) and haloperidol (5mg) randomly assigned to the study participants. They concluded that midazolam had a significantly shorter onset (18.3 +/- 14 minutes) and more rapid time to arousal. Several studies found a significant advantage in combining two or more drugs to achieve maximal sedation. For instance, Battaglia et al[43] and Bieniek et al[44] documented superior efficacy and similar side effect profile for a combination haloperidol and lorazepam versus either drug alone.

Despite the proven efficacy and safety record of neuroleptics and benzodiazepines, they require at least 10-15 minutes for sedation. EXD victims may not have minutes to spare as they continue to struggle against law enforcement or physical restraints in a state of hyperthermia and metabolic acidosis. With the particulars of EXD in mind, we propose intramuscular ketamine as an alternative sedating agent worthy of consideration. It is a drug that can be administered IM (4-5 mg/kg/dose; onset of action: 3-4 minutes) or IV (1-2 mg/kg/dose; onset of action: 30 seconds), does not require endotracheal intubation, and reliably produces rapid analgesia, sedation, and amnesia via direct action on the cortex and limbic system. The use of ketamine for procedural sedation in the pediatric ED and rural operating rooms is popular and has a proven record of efficacy and safety.[45] With 25 years surgical experience in the South Pacific, Reich et al[46] documented that ketamine was effectively used to sedate 866 unmonitored patients without serious complications.

Adult data on ketamine use in the ED is sparse, but one recent literature review by Strayer et al[47] attempted to determine ketamine's adverse effect profile when used for procedural sedation. The analysis revealed that IM ketamine reliably produced adequate sedation to facilitate painful procedures with few side effects. Emergence phenomenon was documented in 10-20% of patients; but if ketamine was administered with a rapidly metabolized benzodiazepine (i.e. midazolam), then the effects were reduced significantly. In a unique retrospective study of 11 combative trauma patients, Melamed et al[48] found that sedation with ketamine, with or without midazolam, was effective in all cases. Ketamine was administered intravenously for sedation by prehospital providers during an average transport time of 114 minutes. Although this report is based on a very small sample size, the authors reported no adverse events and suggest that ketamine might be an ideal intervention for the combative patient.

Although concerns about ketamine causing increased intracranial pressure and/or laryngospasm and subsequent airway obstruction have lessened, the agitated EXD victim represents a unique patient group for future analysis of the drug.[45,49] Furthermore, if a catecholamine surge is at least partially responsible for the medical and psychiatric symptoms of EXD victims, then ketamine might actually exacerbate the underlying problem by acting as a mild stimulant of the cardiovascular system.[46] Therefore, ketamine's most lauded characteristic of having no cardiovascular, respiratory or airway protective reflex depression, might also be cause for concern. One could imagine a scenario in which ketamine's rapid and superior sedation might lure the emergency physician into a false sense of security while the EXD patient is quietly decompensating. Perhaps the potential cardiovascular stimulation could be averted by using a β-adrenoreceptor blocker immediately after sedation with ketamine, as suggested by the results of a recent in vitro study using human atrial myocardium.[50] Despite the promise of ketamine, more structured research is needed to establish its safety and efficacy for emergent sedation of the agitated patient.

In addition to adequate sedation, several protective measures must be taken to increase the chances of survival in persons presenting with EXD. Proper management should arrest the catecholamine cascade quickly. Medical evaluation should begin promptly and include basic monitoring (IV access, pulse oximetry and oxygen), radiographs, blood tests and a focused physical exam. As mentioned previously, EXD victims present with autonomic hyperactivity, which often leads to metabolic acidosis, hyperthermia, and rhabdomyolysis. This clinical picture is similar enough to that of malignant hyperthermia (MH) and neuroleptic malignant syndrome (NMS) that dantrolene could be considered as another useful adjunctive therapy. One informative case report described a 25 year old patient with cocaine-excited delirium and severe acidosis who was treated with hyperventilation, passive cooling, sodium bicarbonate and dantrolene.[51] This intervention lead to a swift correction of the acidosis and the patient survived. Dantrolene is a hydantoin derivative that abolishes excitation-contraction coupling of muscle cells by blocking calcium release from intracellular storage in the sacroplasmic reticulum. It has been used successfully for years by anesthesiologists to treat MH and NMS; however, its use in emergent situations is limited by poor water solubility and difficulties in rapidly preparing a suitable solution for IV administration.[52,53]

CONCLUSION

EXD is a unique medical issue characterized by the acute onset of agitation, aggression, distress, and possibly sudden death. While the contribution of restraint, struggle and the use of electrical conduction devices to the cause of death raises controversy, recent research points toward central nervous system dysfunction of dopamine signaling as a cause of the delirium and fatal autonomic dysfunction. Victims of EXD usually die from cardiopulmonary arrest, although the exact cause of such arrest is likely multifactorial and chronic. Unfortunately, an adequate treatment plan has yet

to be established, although rapid sedation, followed closely by external cooling, IV fluids, monitoring, and treatment of potential medical complications is likely critical to decrease morbidity and mortality. Neuroleptics, benzodiazepines and ketamine are among the potent sedating agents that have been proposed to stabilize EXD victims. While there is still much to be discovered about the pathophysiology and treatment, it is hoped that this extensive review will provide both police and medical personnel with the information necessary to recognize and appropriately respond to EXD.

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Primary Cardiac Tumor Identified as the Cause of Seizure

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A 65-year-old woman presented to the emergency department after a seizure. An unexplained bradycardia and heart murmur were detected and an emergency bedside echocardiography was performed. This revealed a mass in the left atrium. The provisional diagnosis of left atrial tumor was later confirmed by formal echocardiography and ultimately by histology. The first presentation of primary cardiac tumors can be misleading and sometimes presents with neurological manifestations. An early echocardiography can be diagnostic and could lead to early surgical intervention with better prognosis. [West J Emerg Med 2011;12(1):84-86.]

CASE REPORT

A 65-year-old woman presented to the emergency department after an episode described by bystanders as a short episode of tonic – clonic activity with a complete loss of consciousness. She had no urinary incontinence and suffered no obvious traumatic injury. She had suffered two similar episodes during the preceding few weeks and at the time of presentation was waiting for further neurologic investigations. On admission the patient was oriented without evidence of postictal confusion. Breathing and oxygenation were normal. A slow and irregular heart rate (HR: 40/min) with a blood pressure of 160/100 mmHg were noted. There was a loud systolic murmur at the apex. Atrial fibrillation was recorded on the electrocardiogram. Glasgow Coma Scale was 15, and there was no lateralization or other neurological abnormality. We carried out a bedside echocardiography with suspicion of valvular disease which might have led to the sudden collapse. On the echocardiography a 35 x 28 mm mobile mass was attached to the interatrial septum and floating inside the left atrium (Figure 1).

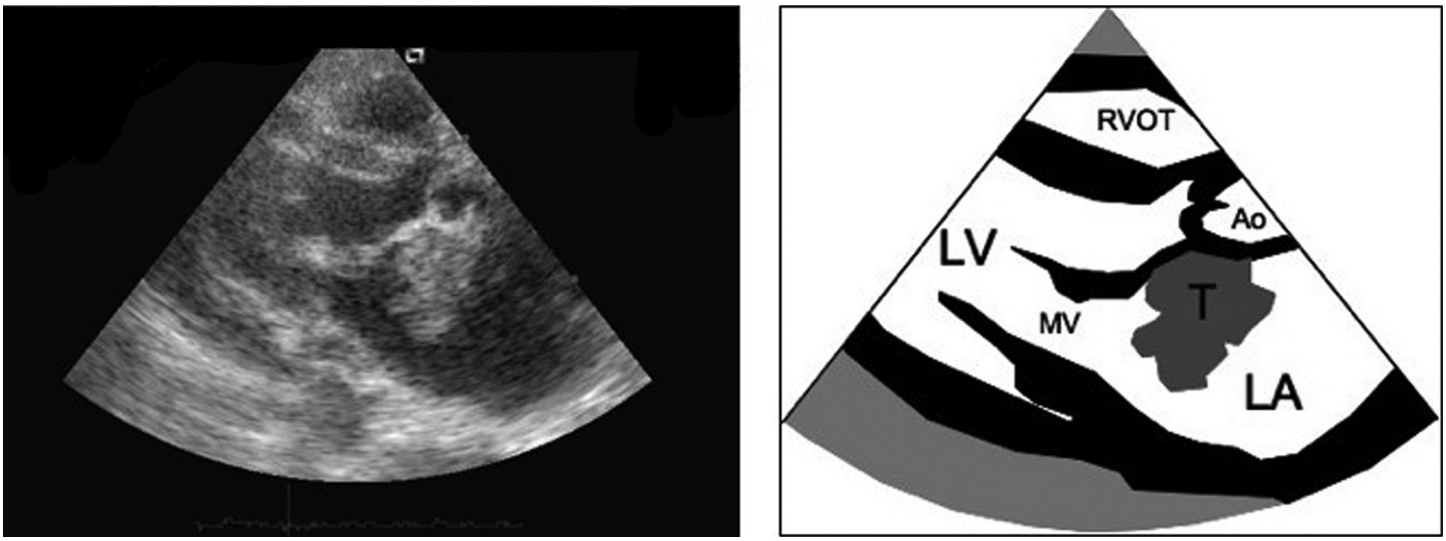

Figure 1. Left atrial tumor on the echocardiography (parasternal long axis view
LA, left atrium; *LV,* left ventricle; *RVOT,* right ventricular outflow tract; *Ao,* aorta; *MV,* mitral valve; *T,* tumor

Following this examination the patient was referred to the cardiothoracic center where further echocardiography raised suspicion for atrial myxoma. Computer tomography (CT) revealed the possibility of multiplex metastatic lesions in the lungs and brain. Unfortunately, the patient's overall status deteriorated before she could undergo cardiac surgery, and she died 12 days after admission. Autopsy verified a 55 x 35 mm atrial tumor with metastases to the pericardium, both lungs and cerebellum. The tumor was classified as mesothelioma by histology.

DISCUSSION

While primary cardiac tumors are rare, their potentially lethal course could sometimes be avoided with timely diagnosis, hence the importance of early diagnosis and adequate surgical treatment. The incidence of primary cardiac tumors in autopsy series ranges from 0.02 – 2.8 / 1000, with a median age of 50 (range: 1 – 81 years) and with a female male ratio of approximately 6:4.[1] About 75% of primary cardiac tumors are benign, most of them being atrial myxoma located in the left atrium.[2] Mesothelioma is usually also benign, but can occasionally be malignant.[2] The clinical symptoms are mostly non-specific and usually originate from one or more of the following main features of these tumors: hemodynamic consequences of the mass obstructing the flow, arrhythmogenic effects due to invasion into the myocardium, distant signs resulting from embolization and systemic non-specific symptoms (anemia, fever, weight loss, etc.).[1,2] Mesothelioma often causes pericardial effusion and tamponade but also can present with pericardial constriction.[3-5] Benign mesothelioma infiltrating the atrioventricular node, causing conduction disturbances and sudden death, has also been described in the literature.[6] Mesothelioma, which infiltrates the atrial wall, can develop an intracavitary pendulous extension mimicking atrial myxoma and can also cause outflow obstruction.[7] Approximately 10% of all primary cardiac tumors can be completely asymptomatic.[8]

Echocardiography is the standard modality for making the diagnosis, although other imaging techniques (magnetic resonance imaging or CT) can be useful for further evaluation. Transthoracic and transoesophageal echocardiography have 95% and 100% sensitivity, respectively.[8] However, echocardiography is usually obtained only if there is a suspected cardiac pathology. It is not a routine investigation after a seizure.

Neurological manifestations of atrial tumors have already been described in prior reports. These are either stroke (usually as a result of embolization from the tumor) or seizures caused mainly by cerebral metastasis.[4] The intracardiac flapping tumor can cause outflow obstruction, which may result in syncope. However, in many cases the syncope is misinterpreted as a seizure.[9] Ekinci et al[10] reviewed 113 cases with neurological presentations of which 12% had seizures and 28% had syncope. The most common neurological symptoms were ischemic stroke (83%), psychiatric presentations (23%) and headache (15%), of which symptoms often overlapped.[10] Sudden hemodynamic collapse has also been described as first presentation of myxoma.[11]

Benign primary cardiac tumors have a good prognosis if diagnosis is established early and surgical excision is successful. Long term (15 – 20 years) survival was reported as 85-92 % .[1] On the other hand, the prognosis of malignant primary cardiac tumors is very poor.[2]

The importance of early diagnosis of primary cardiac tumors and the fact that this could be easily initiated with a simple and non-invasive technique emphasizes the role of focused bedside ultrasound and echocardiography by emergency physicians. The use of echocardiography by non-cardiology medical practitioners is becoming more accepted.[12] Emergency physicians can be trained to perform brief point-of-care focused echocardiographic examinations, which has been shown to be effective in many areas, including assessing left and right ventricular function, recognizing pericardial effusions, and diagnosing valvular abnormalities.[12-14] In our case, the cardiac tumor was discovered by the emergency physician trained in bedside emergency echocardiography, and the patient was immediately referred to the cardiothoracic center where detailed examination was carried out.

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Contagious Weakness in an Elderly Couple with Neurologic Emergencies

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We present an unusual neurologic emergency in an elderly male patient. Given his presentation and risk factors, we presumed the initial symptoms to be secondary to a cerebrovascular accident. As the case evolved, however, it became apparent that a more unusual pathology was present. This case report showcases a rare condition masquerading as a common neurologic emergency. [West J Emerg Med. 2011;12(1):87-89.]

PATIENT PRESENTATION

We present an unusual neurologic emergency. Our patient was an 85-year-old retired Portuguese fisherman who presented with two days of emesis and one day of diplopia, blurry vision and bilateral ptosis. He also complained of difficulty managing secretions and dysphagia but denied headache, stiff neck, numbness, tingling, extremity weakness or difficulty with bowel or bladder control. Past medical history included emphysema and osteoarthritis. He did not take medications and denied drug allergies. Married for 57 years, he lived with his wife. He had quit using alcohol and tobacco 30 years earlier; prior to that, he had smoked three packs a day. He had no recent sick contacts or travel.

Initial vital signs were as follows: temperature of 98.1°F; blood pressure of 163/75mmHg; pulse of 87 beats per minute; respiratory rate of 16 breaths per minute; oxygen saturation of 98% at room temperature. In general, he appeared ill and dysarthric. His head and neck exam revealed pooling of secretions in the posterior oropharynx, bilateral ptosis. No papilledema was noted. He was oriented to person, place and time. His pupils were equal, round, reactive to light and accommodation. Visual fields were full to confrontation. He had limited abduction of both eyes on testing of extraocular movements. He was limited in his ability to protrude his tongue, although it was midline. Shoulder shrug strength, upper extremity strength and lower extremity strength were 5/5 in the proximal and distal muscle groups. We detected no sensory or proprioceptive deficits. Reflexes were 1+ triceps, biceps, and brachioradialis bilaterally and 2+ patellar and ankle jerks bilaterally. Plantars were downgoing bilaterally. Finger-to-nose testing was intact. Gait was not assessed. The remainder of the physical exam was unremarkable.

Laboratory and imaging studies revealed white cell count of 10.9, hemoglobin of 16.2, hematocrit of 49.1 and platelets of 210. The metabolic panel was unremarkable. Chest radiograph showed a possible early right lower lobe infiltrate. Computed tomography (CT) of the head revealed extensive small vessel disease with old bilateral frontoparietal cerebrovascular events. The magnetic resonance imaging (MRI), including diffusion-weighted imaging, did not reveal any evidence of an acute stroke. Lumbar puncture showed one white cell, zero red cells, 31 protein, and 99 glucose. The gram stain was negative as was the acid-fast bacilli stain. India ink preparation was negative.

In the emergency department (ED), primary consideration was given to patient stability. He was mentating without difficulty, had no respiratory distress, and his airway was patent. As his radiographic studies were inconclusive, we considered additional differential possibilities. Given his age, gender and bulbar symptoms, myasthenia gravis figured prominently. Serological studies for acetylcholine receptor antibodies were sent, but results were not immediately

Figure 1. Home canned and processed goods in various stages of spoilage.

Figure 2. Canned tuna, undated. Laboratory confirmed positive for botulinum toxin.

available. Electromyography showed mild slowing of motor and sensory velocities, but these were nonspecific changes. We considered edrophonium testing. This drug acts to inhibit acetylcholinesterase and will improve symptoms in myasthenia gravis by increasing stores of acetylcholine. However, because it may cause marked clinical deterioration and respiratory failure in conditions other than myasthenia, we withheld it, as the patient was stable, and his diagnosis was unclear. He was admitted to the hospital ward for further testing and observation.

Our patient deteriorated precipitously on the first evening of his admission, requiring emergent endotracheal intubation for altered mental status and desaturation. He was completely paralyzed at the time of intubation without medication and was unable to protect his airway, manage secretions, or use the muscles of excursion to facilitate breathing. He remained immobile on the ventilator, despite an early wean of paralytics and sedation. Repeat CT of the head was unchanged.

Five days after his admission, the patient's 90-year-old wife presented with blurry vision and difficulty swallowing. She was directly admitted to the intensive care unit (ICU) for observation. At that point, we were certain of botulism, given the symptoms in both patients. We contacted the state health department, where our request for release of antitoxin from storage at the Los Angeles International Airport was approved, in cooperation with the Centers for Disease Control (CDC). Both patients received the antitoxin the night the wife was admitted. Despite the antitoxin, the patient's wife deteriorated clinically and required mechanical intubation. She had two episodes of cardiac arrest during her ICU stay; however, she was able to be resuscitated and weaned from the ventilator.

We obtained confirmatory testing late in the ICU course. Blood sent for a botulinum toxin murine bioassay was positive for botulinum toxin A. Stool studies were negative for botulinum toxin. Health department staff went to the patient's house and collected home-canned tuna jars and home-canned orange juice. The cans were positive for botulinum toxin A.

Approximately two months after discharge from the ICU, the husband was dependent on ventilatory support via tracheostomy and currently resides in a long-term intensive care facility. His wife has regained some strength but has developed dementia and now resides in a skilled-nursing facility.

DISCUSSION

Botulinum toxin is the most potent biologic toxin known. The organism that produces it, *Clostridium botulinum*, is ubiquitous in the soil. Although several routes of poisoning exist, including wound botulism, which has seen a new emergence in the United States (U.S.) due to intravenous heroin use, and infant botulism via ingestion of spores in a naïve digestive tract, our discussion here focuses on foodborne botulism. Foodborne botulism is caused by the ingestion of preformed toxin that has been expressed by organisms infecting improperly preserved foods. There are seven distinct subtypes, but only A, B, E, and F cause disease in humans. The toxin is inactivated by heating at 85°C for five minutes.[1] It is unlikely that our two elderly patients followed these guidelines.

Symptoms begin anywhere from two hours to eight days after ingestion of the toxin, with most patients presenting within 36 hours. Patients present with a classic descending weakness that begins with dysphagia, diplopia and dysarthria. Ptosis, gaze paralysis and facial paralysis are often noted. Motor paralysis progresses from the upper to the lower limbs. In severe poisonings, the diaphragm and intercostals muscles can be affected, causing asphyxiation without ventilator support. Additional findings can include dry mouth,

constipation, and dilated pupils. In foodborne botulism, gastrointestinal complaints of nausea and vomiting can occasionally occur prior to neurologic findings. Mental status is unaffected, as are sensory findings. Fever is uncommon.[2]

ED care is supportive and may include intubation and, in extreme cases, mechanical ventilation. Additional measures include early notification of public health officials to facilitate antitoxin administration and limit ongoing exposures to other potential victims of the contaminated source.

Fewer than 200 cases of all types of botulism are reported in the U.S. annually. Of these, usually 10 to 30 outbreaks occur each year due to improperly processed foods in the home. In the U.S., clinically significant disease is almost universally attributed to subtypes A, B and E. The trivalent antitoxin contains elements to counteract all of these.[3] As of March 19, 2010, the CDC has released a heptavalent botulism antitoxin under an investigational drug protocol. It is now the only antitoxin available in the U.S. for naturally occurring non-infant botulism.[6]

Early administration of antitoxin lends to the most favorable outcomes. Botulinum toxin causes a flaccid paralysis by blocking release of acetylcholine-containing vesicles within the terminal membrane of the motor neuron. This binding at the neuromuscular junction is irreversible; once the condition has progressed to paralysis, improvement will not occur until new motor axons develop to innervate paralyzed muscle fibers.[2] Antitoxin will stop disease progression by preventing the serum toxin from binding but will not improve motor function in the affected nerve terminals. Once a patient has become ventilator dependent, it will take months until clinical resolution. Complications such as pneumonia, decubitus ulcers and malnutrition are the rule.

The botulism antitoxin is a horse-serum derived product, so significant allergic reactions are likely to occur; 9% of people may have severe reactions.[1] For our patients, we were fortunate to have an allergist available to assist with skin testing prior to antitoxin administration. The antitoxin kit includes instructions and doses for skin testing, so this can be done without the assistance of a specialist, if necessary.

Botulism antitoxin is not routinely stocked in EDs by expert consensus guidelines; it is only accessible via state health departments, whose staffs are able to obtain the antitoxin through the CDC.[4] Antitoxin is stocked in strategic prepositioned areas throughout the U.S. The exception to this is the state of Alaska, where the most common botulism poisoning is type E. Trivalent antitoxin is stocked in many municipalities where this poisoning is common, due to native cultural food practices and processing of whale and seal blubbers. Native village health assistants have been trained to recognize the classic pentad of botulism, specifically "nausea and vomiting, dysphagia, diplopia, dry mouth, and dilated and fixed pupils," and to administer the antitoxin in consultation with state health officials.[5]

The differential diagnosis for acute weakness in the elderly is broad, and a detailed review is beyond the scope of this article. In this patient population, consideration must be given to etiologies as varied as cerebrovascular accidents (CVA), intracranial mass lesions, dementia, chronic subdural hematomas, depression, adverse drug reactions, thyroid derangements and ingestions. Specific causes of descending paralysis may include myasthenia gravis, pontine cerebrovascular accidents, botulism and Lambert-Eaton syndrome.

These disease processes may be somewhat complicated to distinguish. Myasthenia gravis may be confirmed with symptom improvement after the administration of an acetylcholinesterase inhibitor, such as edrophonium. Brain imaging with CT or MRI would likely be abnormal with a pontine CVA. In Lambert-Eaton syndrome, patients will show an improvement in weakness with repetition of exercise or movement. In fact, the Lambert sign, where grip strength will increase progressively throughout the duration of a hand squeeze, is pathognomic. Botulism can be confirmed with toxin assays; however, because the results will be unavailable in the ED, a careful history must be obtained should this diagnosis be considered.

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Differential Use of Diagnostic Ultrasound in U.S. Emergency Departments by Time of Day

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Background: Survey data over the last several decades suggests that emergency department (ED) access to diagnostic ultrasound performed by the radiology department is unreliable, particularly outside of regular business hours.

Objective: To evaluate the association between the time of day of patient presentation and the use of diagnostic ultrasound services in United States (U.S.) EDs.

Methods: This was a cross-sectional study of ED patient visits using the National Hospital Ambulatory Medical Care Survey for the years 2003 to 2005. Our main outcome measure was the use of diagnostic ultrasound during the ED patient visit as abstracted from the medical record. We performed multivariate analyses to identify any association between ultrasound use and time of presentation for all patients, as well as for two subgroups who are more likely to need ultrasound as part of their routine workup: patients at risk of deep venous thrombosis, and patients at risk for ectopic pregnancy.

Results: During the three-year period, we analyzed 110,447 patient encounters, representing 39 million national visits. Of all ED visits, 2.6% received diagnostic ultrasound. Presenting to the ED "off hours" (defined as Monday through Friday 7PM to 7AM and weekends) was associated with a lower rate of ultrasound use independent of potential confounders (odds ratio [OR] 0.73, 95% confidence interval [CI]: 0.65 - 0.82). Patients at increased risk of deep venous thrombosis who presented to the ED during "off hours" were also less likely to undergo diagnostic ultrasound (OR 0.34, 95% CI: 0.15 - 0.79). Similarly, patients at increased risk of ectopic pregnancy received fewer diagnostic ultrasounds during "off hours" (OR 0.56, 95% CI 0.35 - 0.91).

Conclusion: In U.S. EDs, ultrasound use was lower during "off hours," even among patient populations where its use would be strongly indicated. [West J Emerg Med. 2011;12(1):90-95.]

INTRODUCTION

Important concerns are emerging regarding disparities in processes and outcomes of care depending on the time of day or day of week a patient seeks healthcare; poorer outcomes among weekend or evening presentations have been reported for a wide range of conditions including myocardial infarction, gastrointestinal bleeding, and stroke.[1-9] Potential explanations for this disparity have focused on decreased availability of resources, expertise, and diagnostics on weekends and evenings compared with weekdays. [1-9] The

emergency department (ED) is a clinical environment subject to variations in access to resources, expertise, and diagnostics at different times during the week due to the continuous nature of its operation.

Ultrasonography is a diagnostic resource that has increasingly been used in the emergent diagnosis and management for a wide variety of conditions during the past two decades, with findings that led the American College of Emergency Physicians to issue resolutions calling for 24-hour availability of ultrasonography for ED patients.[10,11] Yet, survey data during subsequent years suggests that ED access to radiology department ultrasonography continues to be unreliable, particularly outside of regular business hours.[10-18]

The objective of this study was to conduct the first national quantitative evaluation of ED ultrasound use to measure the extent of any disparity in use attributable to arrival time. We hypothesized that patients who present to EDs on evenings and weekends would undergo ultrasound examination at a lower rate compared with similar patients who present during regular business hours, and that this difference would persist even among patients with conditions for which ultrasound is strongly indicated.

METHODS

Study Design

This cross-sectional study explores the relationship between ED arrival time and ultrasound use using the National Hospital Ambulatory Medical Care Survey (NHAMCS). NHAMCS is an annual cross-sectional survey of ED visits in the United States (U.S.) The survey, conducted by the National Center for Health Statistics, is a four-stage probability sample survey of visits to general and short-stay hospitals, excluding federal, military and Veterans Affairs hospitals. A detailed description of the NHAMCS methodology is available from the National Center for Health Statistics.[19] We conducted the study with the publicly available dataset from NCHS; it was exempt from review by the University of California, San Francisco Human Subjects Committee.

Study Sample

To examine variation in ED ultrasound use, we used all patient data from the 2003-2005 NHAMCS ED surveys. We defined two *a priori* subgroups for secondary analyses: patients with increased risk of deep venous thrombosis (DVT), and patients with increased risk of ectopic pregnancy. We identified patients at increased risk of DVT as those who presented with a primary or secondary complaint (using reason for visit classification - RVC) of unilateral leg swelling (1920.5). We defined patients at increased risk of ectopic pregnancy as those who presented with either primary or secondary complaint of pain during pregnancy (RVC - 1790.1), or spotting/bleeding during pregnancy (RVC - 1709.2). Because many patients are unaware they are pregnant upon arrival to an ED, we also defined patients at increased risk of ectopic pregnancy as those who had International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9) codes at discharge for threatened abortion/hemorrhage in early pregnancy (640), or unspecified complications of pregnancy (646.8, 646.9).

Outcome Variable

The primary outcome variable was the use of diagnostic ultrasound during the ED visit. NHAMCS ultrasound use is based on medical record documentation, and not billing information. (Service Record: 1-53842273: communication with Center for Disease Control ambulatory and hospital statistics division). Use thus included both studies performed by the department of radiology as well as those performed by a non-radiologist (i.e. emergency physician or obstetrics/gynecologist).

Predictor Variables

The primary predictor variable was arrival time to the ED. Arrival time was divided into "regular hours" defined as Monday through Friday 7AM to 7 PM, and "off hours" defined as Monday through Friday nights 7 PM to 7 AM and weekends.

We obtained secondary predictors from predefined NHAMCS variables, including patient characteristics and hospital characteristics. Patient characteristics included both demographics (age, sex, race/ethnicity, method of payment), and clinical data (triage score, triage vital signs, triage pain score). Hospital characteristics included region, hospital ownership type, and Standard Metropolitan Statistical Area (SMSA). Region (Northeast, Midwest, South, and West), and SMSA categories represent standardized geographic divisions defined by the U.S. Census Bureau.[19]

Data Analysis

We used the weights, strata, and primary sampling unit design variables provided by NHAMCS for all analyses. Due to the large sample size, we included all potential predictors in the survey multivariate logistic regression models. To test the regression model, we used a goodness-of-fit test for survey data, analogous to the Hosmer-Lemeshow statistic for independent data. All odds ratios are presented with 95% confidence intervals. We performed all analyses using STATA statistical software (Version 10, StataCorp LP, College Station, Texas). P values less than 0.05 were considered statistically significant.

RESULTS

During the three survey years (2003-2005), NHAMCS collected data on 110,447 encounters, representing an estimated 339 million national visits to EDs. Table 1 shows the overall patient and hospital characteristics of the study sample divided into "regular hours" and "off hours."

Table 1. Comparison of patient and hospital characteristics of emergency department (ED) visits between "regular hours" (Monday-Friday 0700-1900) and "off hours" (Monday-Friday 1900-0700 and weekends)

		ED visits during "regular hours" in estimated millions (% of total) Total estimate =149 million (actual n visits analyzed =48,771)	ED visits during "off hours" in estimated millions (% of total) Total estimate = 190 million (actual n visits analyzed =61,676)
Patient Characteristics			
Female		81 (54%)	102 (54%)
Age	0-17	32.6 (22%)	52.4 (28%)
	18-45	62.9 (42%)	81.4 (43%)
	46-65	29.6 (20%)	32.7 (17%)
	66-80	14.9 (10%)	15.5 (8%)
	Over 80	9.1 (6%)	8.3 (4%)
Race	White only	111 (75%)	143 (75%)
	Black/African American	33.4 (22%)	41.5 (21.8%)
	Asian	2.7 (1.8%)	3.6 (1.9%)
	Native Hawaiian/ other Pacific Islander	0.58 (0.4%)	0.85 (0.4%)
	American Indian/ Alaskan native	0.94 (0.6%)	1.2 (0.6%)
	More than one race reported	0.35 (0.2%)	0.5 (0.3%)
Ethnicity	Not Hispanic	130 (87%)	164 (86%)
	Hispanic	19.3 (13%)	26.4 (14%)
Method of Payment	Private insurance	50.4 (34%)	69.9 (37%)
	Medicare	25.5 (17%)	26.0 (14%)
	Medicaid/SCHIP	33.1 (23%)	44.4 (24%)
	Other	38.3% (26%)	47.9 (25%)
Triage Score	Immediate	17.4 (12%)	24.7 (13%)
	1-14 minutes	19.2 (13%)	23.6 (12%)
	15-60 minutes	52.7 (35%)	67.5 (35%)
	> 1-2 hours	31.3 (21%)	39.4 (21%)
	> 2-24 hours	20 (13%)	24.4 (13%)
	No triage	1.1 (1%)	1.3 (1%)
	Unknown	7.3 (5%)	9.6 (5%)
Hospital Characteristics			
Region	Northeast	31.4 (21%)	36.9 (19%)
	Midwest	35.3 (24%)	45.4 (24%)
	South	56.5 (38%)	73.5 (39%)
	West	25.7 (17%)	34.7 (16%)
Metropolitan Statistical Area	Yes	126 (84%)	160 (84%)
	No	23.1 (17%)	30.1 (16%)
Ownership	Voluntary non-profit	109 (73%)	139 (73%)
	Government, non-federal	25.9 (17%)	33.6 (18%)
	Proprietary	14.1 (10%)	18.2 (9%)
Population Characteristics			
Defined at risk for deep vein thrombosis		0.49 (0.33%)	0.58 (0.30%)
Defined at risk for ectopic pregnancy		1.2 (0.8%)	1.5 (0.8%)

SCHIP, State Children's Health Insurance Program

Table 2. Association between arrival time and ultrasound use in the emergency department (ED) overall and in patients at risk of deep venous thrombosis (DVT) or ectopic pregnancy

	Adjusted Model† OR (95% CI)
Arrival Time to ED	
Regular hours	Reference
Off hours	0.73 (0.65,0.82)
Off Hours Arrival Time to ED	
Regular hours	Reference
Weekend days (Saturday and Sunday, 0700-1900)	0.78 (0.65, 0.94)
Week nights (Monday through Friday, 1900-0700)	0.72 (0.62, 0.83)
Weekend nights (Saturday and Sunday, 1900-0700)	0.67 (0.53,0.85)
Patients at Risk of DVT	
Regular hours	Reference
Off hours	0.34 (0.15,0.79)
Patients at Risk of Ectopic Pregnancy	
Regular hours	Reference
Off hours	0.56 (0.35, 0.91)

†This model adjusted for year, age, sex (except ectopic model), race, ethnicity, payment method, triage score, vital signs, pain level, hospital region, metropolitain statistical area, and ownership.
OR, odds ratio; *CI*, confidence interval

The distribution of "regular hours" and "off hours" visit characteristics is similar across all patient and hospital categories (Table 1). Additional summary data has been reported previously.[20]

In multivariable analysis (Table 2), presenting to the ED during "off hours" was associated with a lower rate of ultrasound use independent of other potential confounders (odds ratio [OR] 0.73, 95% confidence interval [CI]: 0.65 - 0.82, $p < 0.001$). All subcategories of "off hours" times were associated with a significant decrease in use compared with "regular hours" (Table 2). The goodness-of-fit test for survey data indicated acceptable fit ($p = 0.29$).

Table 2 also shows the association for "off hours" ultrasound use in our two subpopulations. For the population at increased risk of DVT, presenting to the ED during "off hours" yielded an adjusted odds ratio of undergoing an ultrasound of 0.34 (95% CI: 0.15 - 0.79, $p = 0.012$). For the population at increased risk of ectopic pregnancy, the adjusted odds of undergoing ultrasound during "off hours" was 0.56 (95% CI 0.35, 0.91, $p = 0.02$).

DISCUSSION

Our principal finding was that in U.S. EDs, the overall use of ultrasound was lower during "off hours" compared to "regular hours." This finding persisted in subgroups at increased risk for DVT and ectopic pregnancy.

Ultrasound is critical to the evaluation of a wide variety of conditions managed in the ED and is considered the imaging modality of choice for DVT and ectopic pregnancy.[21-26] Over the last two decades, expanding patient volume, downsizing of hospital capacity, and an increase in the number of uninsured patients have contributed to growing ED crowding.[27-30] Holding patients overnight in the ED for ultrasound studies to be completed during regular hours of operation, a common practice, has become increasingly untenable. Rather than finding an increase in access to ultrasound services, a recent ED survey noted that there was a perception of a persistent lack of access to this technology.[12] In fact, strategies are commonly used to work around the lack of ultrasound availability in "off hours," such as initiating anticoagulant therapy for presumed DVT without an ultrasound diagnosis and discharging patients with suspected ectopic pregnancy prior to pelvic ultrasound.[31] However, inappropriate therapy or delays in diagnosis can lead to substantial morbidity in both conditions.[21, 31-37]

Our results quantify the difference in use of diagnostic ultrasound by time of presentation in U.S. EDs. This type of disparity is likely to play a role in differential health outcomes based on patients' time of presentation, although our findings cannot directly address this topic. While further study is required in the area of assessing the patient outcomes with regard to this disparity, the decision to obtain an ultrasound should not depend on the time of day or day of the week that the patient arrives in the ED.

LIMITATIONS

NHAMCS offers limited clinical details about each patient encounter. As a result, we were unable to evaluate whether the use of ultrasound was appropriate based on patient presentation. Thus, it is possible that our overall results reflect over-use of ultrasound during "regular hours." To address this concern, we analyzed two *a priori* subgroups that more likely presented with a problem for which ultrasound is strongly indicated in the evaluation. Among these subgroups, the association persists. Although this subgroup approach could lead to misclassification bias, such misclassification would almost certainly be non-differential, which would bias the results towards the null.

CONCLUSION

Our results support the hypothesis that there is a difference in the use of diagnostic ultrasound based on time and day of presentation and this disparity may play a role in differential health outcomes. In practice settings where access to diagnostic ultrasound services is limited, increased training and placement of ED physicians who are

credentialed in the use of limited ultrasonography may help to address this disparity.

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Variable Access to Immediate Bedside Ultrasound in the Emergency Department

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Objective: Use of bedside emergency department (ED) ultrasound has become increasingly important for the clinical practice of emergency medicine (EM). We sought to evaluate differences in the availability of immediate bedside ultrasound based on basic ED characteristics and physician staffing.

Methods: We surveyed ED directors in all 351 EDs in Colorado, Georgia, Massachusetts, and Oregon between January and April 2009. We assessed access to bedside ED ultrasound by the question: "Is bedside ultrasound available immediately in the ED?" ED characteristics included location, visit volume, admission rate, percent uninsured, total emergency physician full-time equivalents and proportion of EM board-certified (BC) or EM board-eligible (BE) physicians. Data analysis used chi-square tests and multivariable logistical regression to compare differences in access to bedside ED ultrasound by ED characteristics and staffing.

Results: We received complete responses from 298 (85%) EDs. Immediate access to bedside ultrasound was available in 175 (59%) EDs. ED characteristics associated with access to bedside ultrasound were: location (39% for rural vs. 71% for urban, $P<0.001$); visit volume (34% for EDs with low volume [<1 patient/hour] vs. 79% for EDs with high volume [≥3 patients/hour], $P<0.001$); admission rate (39% for EDs with low [0-10%] admission rates vs. 84% for EDs with high [>20%] rates, $P<0.001$); and EM BC/BE physicians (26% for EDs with a low percentage [0-20%] vs.74% for EDs with a high percentage [≥80%], $P<0.001$).

Conclusion: U.S. EDs differ significantly in their access to immediate bedside ultrasound. Smaller, rural EDs and those staffed by fewer EM BC/BE physicians more frequently lacked access to immediate bedside ultrasound in the ED. [West J Emerg Med. 2011;12(1):96-99.]

INTRODUCTION

Bedside ultrasonography has become an integral part of emergency department (ED) care and an increasingly important tool for emergency physicians. It provides rapid, real time information that assists in patient care and clinical decision-making.[1-4] Because of its demonstrated value, ED ultrasound has been integrated into emergency medicine (EM) residency training and has become a standard skill set provided by emergency physicians.[5]

Favorable assessments of bedside ED ultrasound come primarily from large, urban, academic centers where training and access to ultrasound is readily available.[13] However, these institutions comprise only a minority of United States (U.S.) EDs, to which most of the U.S. population has limited access.[6,7] Little is known about the use of ED ultrasound outside these urban, academic hospitals. Two prior studies investigating ultrasound use in the community setting have shown decreased use of ED ultrasound compared to the academic

Table 1. Association between emergency department characteristics and access to immediate bedside ultrasound

	Total	Access to Ultrasound		
	N	n	(%)	P value
TOTAL	298	175	59%	--
State				0.001
Colorado	63	42	67%	
Georgia	120	53	44%	
Massachusetts	63	44	70%	
Oregon	52	36	69%	
Urban/Rural status				<0.001
Urban	182	130	71%	
Rural, adjacent to urban	66	20	30%	
Rural, not adjacent to urban	50	25	50%	
ED visit volume (patients/hour)				<0.001
<1	59	20	34%	
1.0-1.9	67	27	40%	
2.0-2.9	31	17	55%	
≥3	141	111	79%	
Admission rate				<0.001
0-10%	69	27	39%	
11-20%	164	99	60%	
>20%	51	43	84%	
Uninsured				0.47
0-15%	91	56	62%	
16-30%	118	73	62%	
>30%	64	34	53%	
Number of physician FTEs				<0.001
0-4	75	25	34%	
5-9	104	54	52%	
≥10	105	86	82%	
EM BC/BE physicians				<0.001
0-20%	72	19	26%	
21-79%	59	33	56%	
≥80%	159	117	74%	

ED, emergency department; *FTE*, full-time equivalents; *EM*, emergency medicine; *BC*, board certified; *BE*, board eligible

Table 2. Independent predictors of immediate access to bedside emergency department ultrasound

	Odds ratio	95% CI
State		
Colorado	**4.55**	1.71 to 12.05
Georgia	Referent	
Massachusetts	1.01	0.42 to 2.44
Oregon	**4.82**	1.74 to 13.38
Urban Influence		
Urban	Referent	
Rural, adjacent to urban	**0.40**	0.17 to 0.99
Rural, not adjacent to urban	0.81	0.29 to 2.25
ED visit volume (patients/hour)		
<1	0.15	0.04 to 0.52
1.0-1.9	**0.25**	0.10 to 0.64
2.0-2.9	**0.30**	0.11 to 0.81
≥3	Referent	
Admission rate		
0-10%	Referent	
11-20%	1.80	0.86 to 3.75
>20%	**3.96**	1.32 to 11.84
Uninsured		
0-15%	Referent	
16-30%	1.33	0.62 to 2.82
>30%	1.22	0.52 to 2.85
EM BC/BE physicians		
0-20%	0.42	0.16 to 1.08
21-79%	0.90	0.42 to 1.93
≥80%	Referent	

Bolded text denotes $P < 0.05$. Number of physician full-time equivalents not included in the model due to collinearity with visit volume.
CI, confidence interval; *ED*, emergency department; *EM*, emergency medicine; *BC*, board certified; *BE*, board eligible

setting, but neither of these studies looked specifically at the effect of rural location on ED ultrasound use.[11,12] Further understanding of ED ultrasound use in the general community, particularly in small, rural EDs, would help educators to target ultrasound training and outreach, as well as improve access to high quality emergency care in all EDs.

In this study, we sought to evaluate overall access to immediate bedside ED ultrasound and differences in access based on basic ED characteristics and physician staffing. We hypothesized that small, rural EDs and those with fewer EM board-certified (BC) or board-eligible (BE) physicians would have less access to bedside ED ultrasound.

METHODS

We conducted a survey of physician or nurse directors of all 351 EDs in Colorado, Georgia, Massachusetts and Oregon between January and April of 2009. Each state investigator's Institutional Review Board approved the study with a waiver of written informed consent.

We used the 2007 version of the National Emergency Department Inventories (NEDI)-USA database to obtain a comprehensive list of all nonfederal U.S. hospitals with EDs in the four included states (n=351). Methods for derivation of NEDI-USA have been previously described.[6] Briefly, NEDI-

USA combines data from three sources: Verispan Marketing Group's Hospital Market Profiling Solution Database (Yardley, PA), the American Hospital Association Annual Survey of the Hospitals (Chicago, IL), and information collected independently by Emergency Medicine Network (Boston, MA) staff. EDs were defined as emergency care facilities open 24 hours per day, seven days per week and available for use by the general public. We excluded federal hospitals (e.g. Veterans Affairs and Indian Health Service hospitals), military hospitals and college infirmaries as these are not available through NEDI-USA.

We mailed the survey to ED directors three times over a two-month period. We contacted non-responders and those with partial or incomplete responses by telephone for completion. The survey assessed access to bedside ED ultrasound by the following question: "Is bedside ultrasound available immediately in the ED?"

We classified ED location as urban and rural (adjacent to urban or not adjacent to urban) using the county-based 2003 urban influence codes (www.usda.gov). Other ED characteristics included in the survey were number of ED patients seen per hour (calculated from annual visit volume), and hospital admission rate. Physician staffing characteristics included total emergency physician full-time equivalents and proportion of EM BC/BE physicians by the American Board of Emergency Medicine, American Osteopathic Board of Emergency Medicine, or the American Board of Pediatrics (Pediatric EM).

Data Analysis

We performed statistical analyses using Stata 10.1 (StataCorp, College Station, TX) and summarized data using basic descriptive statistics. We performed the univariable analyses using chi-square tests to compare differences in access to bedside ED ultrasound by ED characteristics and physician staffing. All p values were two-tailed, with $p<0.05$ considered statistically significant. We then performed multivariable logistical regression to evaluate independent predictors of access to ED bedside ultrasound with results reported as odds ratios (ORs) with 95% confidence intervals (CIs). We included all ED characteristics obtained from the survey, regardless of significance in univariable testing, in the multivariable model.

RESULTS

We obtained complete responses from 298 (85%) of the 351 EDs in the four participating states. The missing 15% was the result of not receiving a completed survey or from not having phone calls returned or answered from ED directors. Overall, 175 (59%; 95%CI, 53-64) had immediate access to bedside ED ultrasound. The tables show access to ultrasound by ED characteristics and physician staffing.

Massachusetts had the highest bedside ED ultrasound access, while Georgia had the lowest. ED characteristics associated with less access to bedside ED ultrasound were: rural location, lower visit volume and lower admission rate. ED physician staffing associated with less access to bedside ED ultrasound were: lower emergency physician full-time equivalents and lower percentage of EM BC/BE physicians.

In the multivariable logistic regression, independent predictors of access to bedside ED ultrasound were ED location, visit volume and admission rate. Rural (adjacent to urban) EDs had less access to ultrasound (OR 0.40 [95%CI, 0.17-0.99]) than urban EDs. Compared to EDs with ≥3 patients per hour, EDs with lower visit volumes had less access to ultrasound (OR 0.15 [95%CI, 0.04-0.52] for <1 patient per hour; OR 0.25 [95%CI, 0.10-0.64] for 1-1.9 patients per hour; and OR 0.30 [95%CI, 0.11-0.81] for 2.0-2.9 patients per hour). Additionally, EDs with admission rates >20% had more access to ultrasound (OR 3.96 [95%CI, 1.32-11.84]) than those with admission rates of ≤10%. After adjusting for ED characteristics, physician staffing was not independently associated with access to bedside ED ultrasound.

DISCUSSION

In a four-state study, with 85% participation, we found that immediate access to bedside ED ultrasound was available in only 59% of EDs. Availability varied significantly by state and by ED characteristics; smaller, rural EDs and those with lower admission rates had significantly less access to bedside ED ultrasound. EDs staffed with a smaller percentage of EM BC/BE physicians had less access to bedside ED ultrasound, although confounding by ED location and visit volume appeared to account for this difference.

These findings have important implications for access to high-quality emergency care and patient safety, especially in smaller, rural EDs. The value of bedside ED ultrasound has been well established in the literature, particularly in situations that require immediate medical decisions and rapid interventions that cannot afford the delay caused by waiting for radiology to perform and interpret an ultrasound study. Specifically, ED ultrasound improves patient safety by decreasing the risks associated with obtaining central venous access, as well as by obviating invasive procedures such as peritoneal lavage, culdocentesis and blind pericardiocentesis.[1,2] Having immediate access to an ED ultrasound machine and appropriately trained providers also improves ED efficiency by providing real-time images without the delay of ordering, performing, and interpreting radiology-performed sonographic studies, which may not be readily available. This efficiency can decrease ED length of stay, help alleviate ED crowding, and most importantly improve emergency care for the patient.[3-4]

While ED characteristics, rather than physician staffing, were most predictive of access to bedside ED ultrasound, our study could not specifically address the reason for these differences. Smaller, rural EDs may not have the patient volume or acuity to financially justify owning a bedside ED ultrasound. Additionally, many smaller, rural EDs are staffed by midlevel providers and non-EM BC/BE physicians.[8-9] These providers

may not be trained to perform and interpret ultrasound studies, limiting the use of bedside ED ultrasound in their practice. Prior studies have demonstrated a shortage of EM BC/BE physicians,[9] which will likely continue for several decades.[10]

Many healthcare providers staffing smaller, rural EDs may not be trained in the use of ultrasound and therefore may not use it to enhance emergency care. Our study suggested that EDs staffed with fewer EM BC/BE physicians had less access to bedside ultrasound. Education and outreach to these small, rural EDs may improve ultrasound training and result in greater access to ultrasound for those patients. Further study is needed to evaluate if increased training and access to bedside ED ultrasound results in a measureable change in quality of emergency care and cost-effectiveness of beside ultrasound for these EDs. While bedside ED ultrasound represents only one important component of ED care, it represents a gap in the ability to provide high-quality emergency care in rural EDs.

LIMITATIONS

The study has several potential limitations. Although we obtained a >80% response rate from all of the EDs in each state, response bias may have affected our results. Specifically, nonresponding EDs may be more likely to have less access to bedside ED ultrasound. However, the urban-rural status and ED visit volumes of nonresponding EDs were similar to those that did respond (data not shown). Additionally, our survey data were limited by reliance on self-reporting. However, ED directors, who were knowledgeable about their ED characteristics and capabilities, were surveyed. However, since survey responses were anonymous, we cannot know if the ED director completed the survey alone or required assistance. Finally, the survey used a single question to evaluate access to bedside ED ultrasound and did not ask about reasons for lack of access nor its actual use. We assumed that immediate access to bedside ED ultrasound was a valid marker for use by ED providers. However, the availability of immediate bedside ultrasound does not require that the ultrasound be performed by the emergency physician; it is possible that a technician or a radiologist could still perform the study. We plan to build on the present results with additional ultrasound-focused questions in future studies to address some of these limitations.

CONCLUSION

There are significant differences in access to bedside ED ultrasound based on ED characteristics and physician staffing. Smaller, rural EDs and those staffed by fewer EM BC/BE physicians had less access to bedside ED ultrasound. Given the proven use of bedside ultrasound in the evaluation and treatment of ED patients, observed differences in access should encourage EM educators and administrators to focus on the diffusion of bedside ultrasound training and access in both small and rural EDs.

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Accidental Carotid Artery Cannulation Detected by Bedside Ultrasound

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This report highlights the importance of using bedside ultrasound in the emergency department to confirm guide-wire placement when performing central venous catheter placement prior to dilating and cannulating the vessel. [West J Emerg Med. 2011;12(1):100-101.]

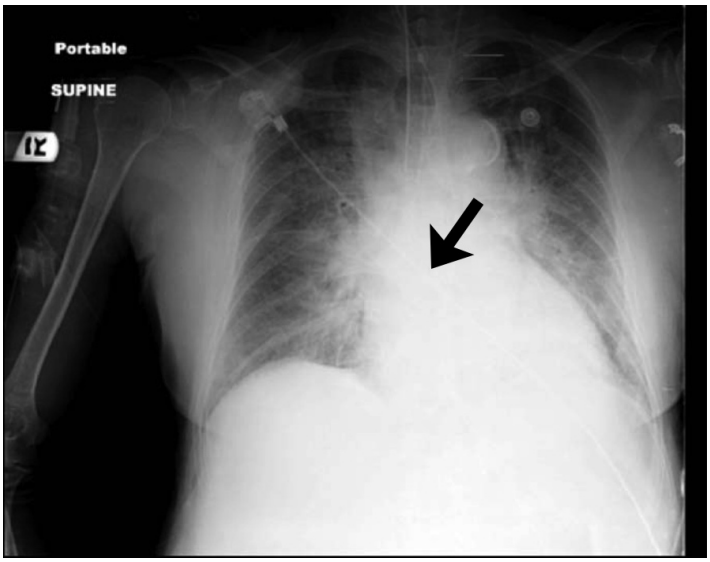

Figure 1. Chest radiograph shows central venous catheter overlying left internal jugular vein traversing the mediastinum.

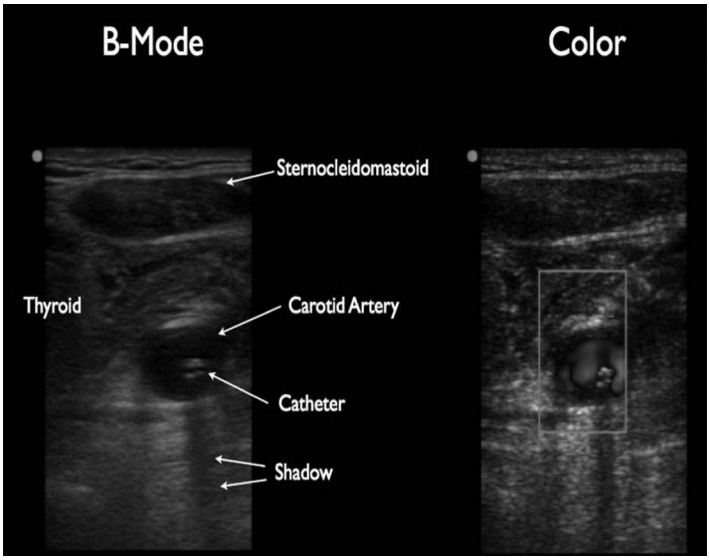

Figure 2. Ultrasound visualization of catheter within the lumen of the left carotid artery with conformation using color flow dropper.

A 73-year-old female was brought to the emergency department for altered level of consciousness. Her blood pressure was 81/50 mmHg, which did not respond to fluid hydration. The right internal jugular (IJ) vein was cannulated with ultrasound guidance for vasoactive administration. A flash-back of dark non-pulsatile blood was obtained before vessel dilation and catheter placement. A portable chest radiograph was performed and read by the staff radiologist as "left IJ catheter is seen overlying the mediastinum likely within the left innominate vein" (Figure 1). Prior to the use of the catheter, the nursing team noted abnormal pulsations from the distal port of central venous catheter (CVC), prompting a bedside ultrasound by the clinician to ensure proper placement (Figure 2). Differentiation of the carotid artery and IJ vein was difficult secondary to the patient's severe volume depletion, but doppler showed pulsatile flow in the cannulated vessel confirming inadvertent arterial placement.

This report highlights the use of using ultrasound to confirm proper placement of a central venous catheters. The IJ vein overlies the carotid artery, making chest radiograph an unreliable test to ensure venous placement. Central venous pressure (CVP) monitoring can confirm venous cannulation, but can be difficult to obtain in many emergency departments. Classic teaching of looking for dark non-pulsatile blood can be

inaccurate in hypotensive and hypoxic patients. Also when a collapsible vein overlies the carotid artery, a through puncture can occur.[1] A prior study demonstrated the use of guidewire visualization with bedside ultrasound as a useful technique to confirm venous placement prior to dilation.[2] We feel that with the fair amount of literature demonstrating improved safety of ultrasound-guidance in CVC, confirming venous placement of either the guidewire or catheter can be a simple addition in hypotensive patients or those that uncertainty exists regarding arterial cannulation.

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Identification of Sonographic B-lines with Linear Transducer Predicts Elevated B-Type Natriuretic Peptide Level

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For editorial comments see p 106.

Objective: This study sought to correlate the presence of pleural-based B-lines seen by emergency department ultrasound performed with the linear transducer with B-type natriuretic peptide (BNP) level in patients with suspected congestive heart failure.

Methods: The study was a prospective convenience sample on adult patients in an academic, urban emergency department with over 100,000 annual patient visits. Adult patients with a BNP level ordered by the treating physician were prospectively enrolled by one of four physicians, blinded to the BNP level. The enrolling physicians included an emergency ultrasound director, two emergency ultrasound fellows, and a senior emergency medicine resident. Bedside ultrasound was performed using a 3-12 MHz linear broadband transducer in four lung fields. The serum BNP level was correlated with bilateral B-lines, defined as three or more comet-tail artifacts arising from the pleural line extending to the far field without a decrease in intensity on the right and left thorax.

Results: Sixty three patients were consented and enrolled during a four-month period. Fifteen patients had the presence of bilateral B-lines. The median BNP in patients with bilateral B-lines was 1560 pg/mL (95% confidence interval (CI) 1141-3706 pg/mL), compared with 538 pg/mL (95% confidence interval 310-1917 pg/mL) in patients without B-lines. The distributions in the two groups differed significantly (p=0.0006). Based on the threshold level of BNP 500 pg/mL, the sensitivity of finding bilateral B-lines on ultrasound was 33.3% (95% CI: 0.19-0.50), and the specificity was 91.7% (95% CI: 0.73-0.99). In addition, bilateral B-lines were absent in all patients with a BNP<100 pg/mL.

Conclusion: The presence of bilateral B-lines identified with the linear probe is associated with significantly higher BNP levels than patients without B-lines. In our patient population, the presence of B-lines was specific but not sensitive for BNP>500. Further research may show that it can be applied to quickly assess patients with undifferentiated dyspnea. [West J Emerg Med. 2011;12(1):102-106.]

INTRODUCTION

In the United States approximately three million people live with congestive heart failure (CHF), and approximately 400,000 new cases are diagnosed each year.[1] Prior studies have demonstrated the benefit of early diagnosis and treatment in decompensated CHF.[2, 3] However, the clinical diagnosis of CHF may be difficult even for expert physicians.[4] The use of serum B-type natriuretic peptide (BNP) coupled with clinical exam significantly improves the diagnostic accuracy in determining acute decompensated CHF. However, the immediate result of this lab test is not generally available.[5]

Bedside physician-performed ultrasound is increasingly available in academic and community emergency departments (ED). Ultrasound of the thorax may show evidence of extravascular lung water present in patients with CHF.[6] Artifacts known as "Lung Rockets," or "B-lines," which represent interstitial fluid seen on the periphery of the pleura have been associated with CHF.[7] Interstitial pulmonary

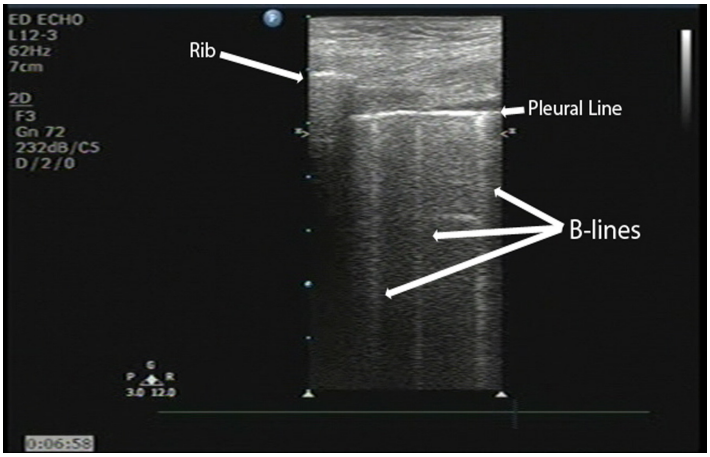


Figure 1. Sonographic B-lines identified with a linear transducer

edema has been reliably detected in multiple studies by chest ultrasound and has been used in the distinction between Chronic Obstructive Pulmonary Disease (COPD) and CHF exacerbation.[7-9] However, the sensitivity, specificity, and correlation of "B-lines" with other objective measures of CHF in the ED setting remains incompletely defined. Prior studies have used curvilinear or micro-convex transducers to identify the presence of B-lines. However, no study has yet to evaluate the ability of linear transducers to explore these findings, despite their widespread use to detect pneumothoracies.

The purpose of this study was to correlate the presence of B-lines using emergency physician-performed chest ultrasound with the linear transducer with BNP level in patients with suspected CHF. We hypothesized that the presence of bilateral B-lines would be associated with a significantly elevated serum BNP in patients with suspected CHF.

METHODS

Study Design

The study was a prospective convenience sample of adult patients presenting to the ED in whom the treating physician ordered a BNP level. The study team was composed of a director of emergency ultrasound, two emergency ultrasound fellows and a senior emergency medicine resident. The study team worked non-clinical shifts in the ED during pre-defined four-hour blocks. Generally, enrollment occurred during the weekday morning or afternoon. During the enrollment periods, the study team was not responsible for direct patient care. The study team member would ask the treating physician if a BNP level had been ordered on any patient. The study did not discriminate whether BNP had been ordered for suspicion of heart failure or other reasons.

Prior to enrolling patients, the study team received goal-directed training in thoracic ultrasound. The training included a one-hour lecture with at least 25 thoracic ultrasound exams reviewed by the ultrasound director. Prior to the study, each of the fellows had completed at least 300 bedside ultrasound exams and the emergency medicine resident had completed approximately 100 ultrasound exams. The ultrasound director had completed over 2,000 bedside ultrasound examinations. The study was approved by human subjects committee at the study institution. Each enrolled patient gave written informed consent.

Study Setting and Population

We conducted the study in the adult ED of an urban Level 1 trauma center with an emergency medicine residency and greater than 100,000 patient visits per year. Patients were included if they were at least 18 years old, and a serum BNP was ordered in the ED. We excluded patients if they were non-English speaking, a prisoner or incapable of informed consent without an available representative.

Study Protocol

After informed consent, we collected baseline data on the patient and a bedside ultrasound was performed. The study investigator was blinded to the serum BNP level, other laboratory values and chest radiograph at the time of the ultrasound examination. However, the study team was not blinded to the patient's appearance, vital signs or other clinical data. Ultrasound was performed with a portable ultrasound scanner (Philips HD-11XE, Andover, MA) using a 3-12 MHz broadband linear transducer in the echocardiography preset with tissue harmonic imaging enabled. Patients were examined lying in the stretcher in the position of comfort. Generally, patients were sitting upright or in a semi-recumbent position. No patient was examined in the full supine position. We investigated four thoracic ultrasound windows: right mid-axillary, right mid-clavicular, left mid-axillary and left mid-clavicular. The probe was placed in a cephalic orientation between the fourth and fifth ribs. The depth settings were adjusted so that the pleural line was placed in the middle of the image. Therefore, the total depth ranged from one patient to another, based on body habitus. All ultrasound examinations were recorded as dynamic images on DVD for later review by the emergency ultrasound director.

Measurements

Prior to the enrollment of patients, the study team defined the presence of multiple B-lines as three or more comet tail artifacts originating from the pleural line, extending to the bottom of the ultrasound screen without a decrease in signal intensity as depicted in Figure 1. Bilateral B-lines were defined when multiple B-lines were present on both the left and right. The study investigator immediately recorded the ultrasound findings on a structured data sheet (see Appendix). The emergency ultrasound director later reviewed these findings to confirm the correct interpretation. During the review, the ultrasound director was blinded to the other data. In a subset of patients, a second credentialed physician, who was blinded to the results of the first ultrasound, repeated the ultrasound measurements.

Table 1. Presence of thoracic B-lines compared to low, intermediate and high B-type natriuretic peptide (BNP) levels.

	BNP > 500pg/mL	BNP 100-500pg/mL	BNP < 100pg/mL	Total
Bilateral B-lines present	13	2	0	15
Bilateral B-lines absent	26	9	13	48
Total	39	11	13	63

Table 2. Presence of B-lines based on B-type natriuretic peptide (BNP) threshold of 500pg/dL.

Thoracic Ultrasound	BNP level > 500pg/mL	BNP level < 500pg/mL	Total
Bilateral B-lines present	13	2	15
Bilateral B-lines absent	26	22	48
Total	39	24	63

Variable	Value	95% confidence interval
Sensitivity	0.33	0.19 to 0.50
Specificity	0.91	0.72 to 0.98
Positive predictive value	0.86	0.59 to 0.98
Negative predictive value	0.45	0.31 to 0.60
Likelihood ratio	4.0	

Data Analysis

We recorded all data in an Access database (Microsoft Corp., Seattle, WA) and performed statistical analysis using SPSS 15.0 (SPSS Inc., Chicago, IL). We determined the median BNP value for the patients with and without bilateral B-lines and calculated 95% confidence interval (CI) of the difference. Initially, a receiver operating characteristic (ROC) curve was planned to analyze the data, but further evaluation of the data concluded that a Mann-Whitney U test would better delineate the significance of the difference in rank order of BNP in those patients with and without bilateral B-lines. At the study institution, a BNP level was considered positive at the threshold limit of 500 pg/mL.

RESULTS

We consented and enrolled 63 patients during a four-month period. Of the 63 patients, the ultrasound director enrolled three, the ultrasound fellows enrolled 34, and the emergency medicine resident enrolled 28. Of the 63 patients, nine had repeat exams by a second sonologist that showed complete agreement with the presence or absence of multiple bilateral B-lines. The mean age was 70 (range 31-90 years); 51% were male. Fifteen of the 63 (24%) had bilateral B-lines.

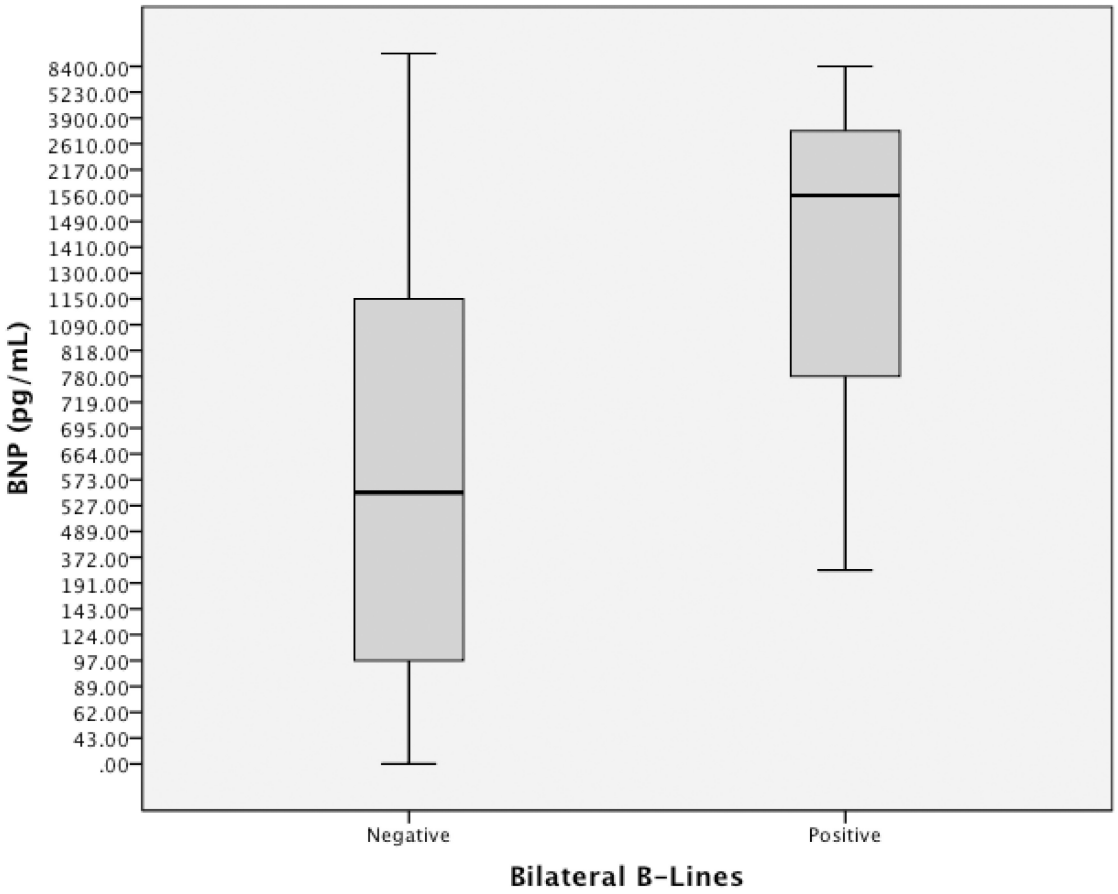
Post-hoc analysis revealed that multiple bilateral B-lines were absent in all patients with a BNP<100 pg/mL. Of the 15 patients with B-lines, only two had a BNP level between the indeterminate range of 100-500 pg/mL (306 and 489). Thirteen of the 15 patients with B-lines had a BNP level greater than 500 pg/mL (Table 1).

The median BNP in patients with bilateral B-lines was 1560 pg/mL (95% CI 1141-3706 pg/mL), compared with 538 pg/mL (95% CI 310-1917 pg/mL) in patients without B-lines. The distributions in the two groups differed significantly (Mann-Whitney U-statistic = 147.5, p=0.0006, two tailed) [Graph]. Based on the threshold level of BNP 500 pg/mL, the sensitivity of finding bilateral B-lines on ultrasound was 33.3% (95% CI: 0.19-0.50), and the specificity was 91.7% (95% CI: 0.73-0.99). The positive predictive value of bilateral B-lines was 0.87 (95% CI: 0.60-0.98) and the negative predictive value was 0.46 (95% CI: 0.31-0.61) [Table 2].

LIMITATIONS

This pilot study was performed on a convenience sample by a small group of trained investigators. Further work is needed to determine if these findings are broadly applicable. The primary limitation of this study is that it only looked at the correlation between bedside ultrasound and BNP. While BNP has been shown to accurately discriminate between CHF and non-CHF causes of dyspnea at certain levels (>500pg/dl or <100pg/dl), it is an imperfect gold standard. Additionally, the small group of trained investigators may limit this study's applicability to other hospital settings. The majority of the patients (58.7%) were enrolled by either emergency ultrasound fellows or the ultrasound director. However, the emergency medicine resident with limited prior ultrasound experience enrolled the remainder of the patients. Future studies will be needed to determine whether inexperienced sonologists are capable of repeating these results.

In this study we consistently used the same linear ultrasound probe with the echo preset and tissue harmonic imaging enabled. The authors believe that these settings best emphasize the B-line artifact. However, some machines will not allow the user to apply a cardiac preset on linear probes. Multiple research studies regarding the detection of a pneumothorax with bedside ultrasound have used the linear probe.[10, 11] While the evaluation of pneumothorax with bedside ultrasound has been included with the recent American College of Emergency Physician Ultrasound Guidelines, many emergency physicians are not familiar with the evaluation of CHF with bedside ultrasound.[12] The evaluation of all types of



Graph. Box-plot of B-type natriuretic peptide (BNP) levels in patients with and without bilateral B-lines. Negative = patients without bilateral B-lines on ultrasound; Positive = patients with the presence of bilateral B-lines on ultrasound.

dyspnea with the linear probe may represent a more natural progression for the average emergency physician.

While the bedside ultrasound was performed within a short amount of time after the serum BNP level was sent to the lab, this study did not specify a cut-off time between the two events, and patients may have been treated in the interim. Prior studies have shown that interstitial edema changes rapidly and dynamically in dialysis patients,[13] and the results of this study do not reflect whether the patients had received any prior treatment. Simultaneous phlebotomy and chest ultrasound may alter results. Additionally, this study did not discriminate whether BNP had been ordered for suspicion of heart failure or other reasons. This study was conducted at a teaching institution where residents at various training levels often order labs. The BNP laboratory tests ordered may not necessarily translate to clinical suspicion of CHF. This fact may explain why the majority of ultrasound exams of patients with elevated levels of BNP did not illustrate the presence of multiple bilateral B-lines.

This study, like most studies with B-lines, did not prevent the sonologist from simultaneously seeing the study subject. This clearly introduces a bias to the enrolling physician, as the investigator is not blind to physical findings and ongoing treatments such as Bi-level Positive Airway Pressure, etc. On the other hand, when this test is applied clinically, this bias is also present and in fact integral to the concept of bedside ultrasound. The additional tools and information that bedside ultrasound provides the treating physician are in fact the crux of its value and importance.

Further research will define the role of bedside ultrasound evaluation of the pleural line to improve immediate diagnostic capability of a focused thoracic exam. Future research may also investigate the effect of B-lines on post-test probability of CHF.

DISCUSSION

This is the first published study to show that the presence of B-lines correlates with higher BNP level in patients using physician-performed ultrasound with a linear probe. This brief exam takes under a minute and is easily learned and reproduced.[14] While earlier literature has identified similar results with curved probes, no prior studies have extensively used the linear probe for this application.[15]

"Comet tails" are reverberation artifacts caused by an air fluid interface that result in a hyper-echoic line that extends

distally from the visceral pleura. Small, tapering hyper-echoic lines that diminish distally are also known as "Z lines" and are often present in healthy individuals. Their presence has been shown to be helpful in ruling out pneumothorax.[16] The more pathologic "B lines" (or "Lung Rockets"), which emanate from the pleural line in the presence of extravascular lung water, create hyper-echoic lines that do not diminish distally.[6] While this study showed correlation between B-lines and BNP, B-lines are not entirely specific to CHF and may be present in other conditions, such as acute respiratory distress syndrome, chronic interstitial lung disease, and high altitude pulmonary edema.

Based on pilot evaluation of several patients, the linear probe with a cardiac setting (with a low dynamic range) enhanced the contrast and clarity of the lung-rocket artifact. However, other researchers have used different probes and settings that may affect the identification of B-lines. For instance, an eight-lung window protocol was used in the studies by Liteplo et al,[15] Volpicelli et al[17] and Lichtensteinet al[6] used a six-window protocol. We also chose to focus on four-lung fields to simplify the examination. In fact, the recent study by Liteplo[15] suggests that the easier two-zone test may suffice for the evaluation of CHF. It does appear that "B-lines" tend to appear more frequently in the dependent portions of the lung, even in healthy patients.

We are unsure why the majority of patients in this study with elevated BNP levels did not exhibit bilateral B-lines. This study introduces a unique problem for the evaluation of B-lines with bedside ultrasound. Although the specificity of the B-lines is similar to other studies, the sensitivity of the test is significantly lower than other studies. In the future we intend to analyze the contribution of systolic and diastolic cardiac dysfunction and the presence of elevated preload to help differentiate these patients.

CONCLUSION

This study showed that the presence of B-lines in patients correlates with increased BNP level while using a linear transducer. The presence of B-lines is specific for an elevated BNP level but not sensitive. Presence or absence of B-lines can be rapidly determined using bedside emergency ultrasound and may aid in diagnosis of patients with suspected CHF.

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Great Tool or Gold Standard? B-Type Natriuretic Peptide and Congestive Heart Failure

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See also p. 102

We would like to thank the contributing authors Manson et al[1] for their article "Identification of Sonographic B-Lines with Linear Transducer Predicts Elevated B-Type Natriuretic Peptide Level."By attempting to validate this easily learned and reproducible ultrasound technique, they have contributed to the growing body of research that suggests bedside thoracic ultrasound can provide rapid point-of-care testing to help guide the diagnosis of congestive heart failure (CHF) in the emergency department (ED).[2]

The rapid diagnosis of CHF in ED patients with undifferentiated dyspnea remains a challenge. Although we have a multitude of clinical information that suggests heart failure, no single aspect of the history, physical examination, echocardiogram, chest radiograph or blood chemistry is specific or sensitive enough to rule in or out the diagnosis.[3] Rather, clinicians must incorporate all of this data into their clinical assessment. Decision scores such as the Framingham and Boston criteria have been developed as systematic ways of weighing such data against the probability of CHF.[4,5]

B-type Natriuretic Peptide (BNP) is an important tool amongst these diagnostic modalities. Multiple studies have demonstrated its effectiveness, particularly its ability to help rule out the diagnosis of CHF when levels are below cutoff values (typically 100). This high negative predictive value is often used in the ED to distinguish dyspnea caused by heart failure from exacerbations of chronic obstructive pulmonary disease.[6]

It is important to remember, however, that BNP is not to be used as a stand-alone test. Multiple factors besides CHF can contribute to an elevated BNP level. These include advanced age, renal failure, acute coronary syndromes and atrial fibrillation.[7,8] BNP levels between 100 and 500 pg/ml fall into a range that are less helpful in excluding or confirming the diagnosis. Therefore, any interpretation of BNP should be made only after the clinician has established the pre-test probability of CHF using all available clinical data.

Just as an isolated BNP value is unacceptable as proof of diagnosis of CHF in clinical practice, so too is its use as a lone diagnostic standard in the research setting. The research submitted by Manson et al. does not attempt to validate bedside ultrasound as a means of differentiating CHF. Rather they test the ability of ultrasound to predict a cutoff value of BNP, a surrogate for the diagnosis. As a result, we are left with a correlation between ultrasound findings and lab values—each one of which must be interpreted with the corresponding patient's clinical presentation if it is to accurately suggest CHF.

If we are to validate such techniques in the future, we must use more accurate methods in establishing the diagnosis of CHF. Because there is no single sufficient diagnostic criterion, the research standard of diagnosing CHF should be the same as those in clinical practice—namely, the judgment of physicians. Prospective studies that include retrospective review by a panel of physicians, considering all presenting signs, symptoms, and diagnostic data of the subject in question, are the most widely accepted and reasonable basis of establishing the diagnosis of CHF. Although such methods are costly and time consuming, they should be considered the criterion standard for further research.

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The Standard of Care: Legal History and Definitions: the Bad and Good News

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The true meaning of the term "the standard of care" is a frequent topic of discussion among emergency physicians as they evaluate and perform care on patients. This article, using legal cases and dictums, reviews the legal history and definitions of the standard of care. The goal is to provide the working physician with a practical and useful model of the standard of care to help guide daily practice. [West J Emerg Med. 2011;12(1):109-112.]

INTRODUCTION

The concept of "the standard of care" is often discussed among physicians, and yet the legal definition of this term is frequently not understood. Emergency physicians are on the front lines of medicine and are frequently involved in medical malpractice cases. It is estimated that 7-17 malpractice claims are filed per 100 physicians every year.[1,2] States vary in the number of these claims that result in payment (Table 1).[3] Thus it is important to know how the legal system defines the standard of care, and to what standards we as physicians are being held. A chronological approach to the evolving definition of the standard of care through legal history will help to understand the current concept and nuances of the term.

Negligence, in general, is legally defined as "the standard of conduct to which one must conform… [and] is that of a reasonable man under like circumstances."[4] In law, medical malpractice is considered a specific area within the general domain of negligence. It requires four conditions (elements) be met for the plaintiff to recover damages. These conditions are: duty; breach of duty; harm; and causation. The second element, breach of duty, is synonymous with the "standard of care." Prior to several important cases in the 1900s, the standard of care was defined by the legal concept of "custom." As quoted in the 1934 case of *Garthe v. Ruppert*, when "certain dangers have been removed by a customary way of doing things safely, this custom may be proved to show that [the one charged with the dereliction] has fallen below the required standard." [5] Put another way, if others in the business are commonly practicing a certain way that eliminates hazards, then this practice can be used to define the standard of care. A jury still needed to decide, however, whether this "custom" was reasonable and whether the deviation from this "custom" was so unreasonable as to cause harm.

EARLY LEGAL CASES: THE BAD NEWS

Two cases changed the legal definition of the standard of care as it is applied in medical malpractice law today. The first case had nothing to do with medicine, but rather a tugboat. The case of *The T.J. Hooper* in 1932 helped to alter how the legal profession thought about custom and the standard of care. In this case, the owner of the tugboat T.J. Hooper was sued for the value of two barges. The tugboat had been caught in a storm and the two barges it was transporting sunk. The

Table 1. Tne worst paid malpractice claims per state per year.

State	Paid claims per 1000 physicians
Louisiana	21.8
Montana	21.6
New Mexico	18.9
Oklahoma	18.3
New York	17.3
Puerto Rico	17.1
South Carolina	16.1
Pennsylvania	15.5
Mississippi	14.2
Kansas	13.9

owners of the barges charged that the T.J. Hooper was unsafe for duty at sea as it did not have a radio receiver to review important storm warnings. In addition, they charged that it was "customary" for tugboats to have this radio receiver. They claimed that if the T.J. Hooper had a radio, they could have been warned of the storm and avoided it. In reviewing the case during appeal, Justice Learned Hand ruled in favor of the barge owners; however, he did not do so based on custom. He indicated that it was not in fact customary for tugboats to be outfitted with the receivers, but that since the practice was reasonable, the owners of the T.J. Hooper could be help liable for damages. He stated, "In most cases reasonable prudence is in fact common prudence; but strictly it is never its measure; a whole calling may have unduly lagged in the adoption of new and available devices. It never may set its own tests, however persuasive be its usages. Courts must in the end say what is required; there are precautions so imperative that even their universal disregard will not excuse their omission."[6] In other words, if there is a practice that is reasonable but not universally "customary" it may still be used as a measure of the standard of care.

The case of *The T.J. Hooper* set the stage for an important trial in medical malpractice that occurred in 1974. In the case of *Helling v. Carey*, the plaintiff (Helling) sued her ophthalmologist (Carey) for the loss of her eyesight due to glaucoma. The defendant won both during the original trial and the appeal, but when the case made it to the Supreme Court of Washington State the verdict was overturned in favor of the plaintiff. During the initial trials, the expert witnesses indicated that as the patient was under the age of 40 and the incidence of glaucoma in this group was only one in 25,000, that it was not the standard to test patients under 40 with tonometry. The Supreme Court decided, however, that the test was inexpensive and harmless, and should have been offered to the patient. Justice Hand's decision in *The T.J. Hooper* case was quoted in the decision.[7]

The case of *Helling v. Carey* set a worrisome precedent for medical malpractice cases. The court essentially ruled that even though the customary practice at the time was followed, the physician was still liable. They cited the case of *The T.J. Hooper* and also referenced a decision by Justice Oliver Wendell Holmes in 1903 that stated ,"what usually is done may be evidence of what ought to be done, but what ought to be done is fixed by a standard of reasonable prudence, whether it usually is complied with or not."[8] These two cases legally established that while great weight is given to customary practices with regard to the standard of care, custom is not the definitive factor in determining negligence. In essence, the two cases suggest that what is commonly done (i.e. custom) may not be enough, and that there are some things that may not be standard, but are still reasonable for the physician to perform. Unfortunately for the physician, these cases suggest it is up to the legal profession and the jury, and not the medical profession, to decide what is "reasonable" and "unreasonable." In fact, subsequent studies have found that *Helling v. Carey* has changed the practice of offering tonometry to all patients with subsequent increase in cost and no change in morbidity.[9] Following the ruling in *Helling v Carey*, there was an outcry from physicians. The medical profession as a whole seemed to be asking, "How much is enough?"

The ruling in *Helling v Carey* prompted state legislatures to pass statutes that defined the standard of care in their jurisdiction. The state of Washington was the first to pass this type of legislation, when they stated that the standard of care is not met when "the defendant or defendants [fail] to exercise that degree of skill, care and learning possessed by other persons in the same profession…"[10]

MODERN CASES: THE GOOD NEWS

The good news for practicing physicians is that in more recent cases there appears to be an effort to ensure that jurors understand that the standard of care does not mean perfection in practice. While old cases in law tend to be more powerful as they have stood the test of time, these more recent cases help show a trend toward keeping jury expectations realistic.

In the 1985 case of *Hall v. Hilbun*, a patient (Mrs. Hall) presented to her physician for abdominal pain. Dr. Hilbun, a general surgeon, was consulted and operated on the patient for a small bowl obstruction. He observed the patient in the recovery room and left for the night. Details from the case indicate that there were abnormal vital signs, and Mrs. Hall had pain throughout the night, but Dr. Hilbun was not notified. She died of respiratory failure in the morning. During the night, Dr. Hilbun had been notified about another patient, but he did not check up on Mrs. Hall. In addition, his orders never indicated for what things he should be called by the nursing staff. Initially he won the case because the testimony of two witnesses that discussed the national standard of care for a surgeon was excluded. On appeal, however, Dr. Hilbun was found liable as their testimony was allowed. Even though the physician lost in this case, the court's discussion was very important in defining the standard of care in the modern era. Chief Justice C.J. Robertson stated:

> *"Medical malpractice is a legal fault by a physician or surgeon. It arises from the failure of a physician to provide the quality of care required by law. When a physician undertakes to treat a patient, he takes on an obligation enforceable at law to use minimally sound medical judgment and render minimally competent care in the course of services he provides. A physician does not guarantee recovery… A competent physician is not liable per se for a mere error of judgment, mistaken diagnosis or the occurrence of an undesirable result."*[11]

In this case, Dr. Hilbun did not provide "minimally

competent care," but the good news from a physician's standpoint is that the law only requires "minimal competence." The care does not even have to be "average," which makes sense; otherwise, 50% of all medical care would be malpractice by definition.

A second case with similar outcomes occurred in 1995. In the case of *McCourt v. Abernathy* the physicians again lost due to their substandard care. Mrs. McCourt presented for several complaints over the course of three days but was found to have an infection on her finger from a pin stick while working in manure. Over the course of these three days, she was seen by Dr. Abernathy and his partner Dr. Clyde who simply cleaned the wound. When she became increasingly ill, they gave her oral antibiotics, but she subsequently became septic. An internist who was consulted diagnosed septicemia, and the patient died despite aggressive care. Again, the physicians acted below the standard of care, but the trial judge gave an important set of instructions to the jury. He stated:

> *"The mere fact that the plaintiff's expert may use a different approach is not considered a deviation from the recognized standard of medical care. Nor is the standard violated because the expert disagrees with a defendant as to what is the best or better approach in treating a patient. Medicine is an inexact science, and generally qualified physicians may differ as to what constitutes a preferable course of treatment. Such differences due to preference…do not amount to malpractice.*
> *I further charge you that the degree of skill and care that a physician must use in diagnosing a condition is that which would be exercised by competent practitioners in the defendant doctors' field of medicine….*
> *Negligence may not be inferred from a bad result. Our law says that a physician is not an insurer of health, and a physician is not required to guarantee results. He undertakes only to meet the standard of skill possessed generally by others practicing in his field under similar circumstances."*[12]

Again, the judge re-enforced that the care provided by a physician be minimally competent, may differ from the care of other physicians and that a bad outcome does not mean that the standard of care was not met.

A final case that helped define the modern definition of the standard of care is *Johnston v St. Francis Medical Center* from 2001.[13] In this case, a 79-year-old male who presented with abdominal complaints was evaluated with radiographs and labs, but his examination was equivocal. Two physicians examined him during the course of the day and found him to be in mild distress. Additional studies to include computed tomography and ultrasound were ordered, but the patient became hypotensive and was sent to the intensive care unit (ICU). The ICU physician thought he might have an aortic aneurysm, which was confirmed during laparotomy. The patient died in the operating room. The plaintiffs argued that the physicians should have diagnosed the aneurysm earlier. All of the experts, except one, in the case indicated this was a difficult diagnosis. The court ruled in favor of the physicians. More importantly however; the court made it clear that while the aneurysm was obvious on radiograph and labs once the diagnosis was made, hindsight can not be used for evaluating the conduct and judgment of the physician. In this case the diagnosis of aneurysm was "possible" but difficult enough that missing the diagnosis did not amount to not providing the standard of care. This is in sharp contrast to the prior case of *Helling v. Carey*.

CLINICAL PRACTICE GUIDELINES AND THE STANDARD OF CARE

A brief discussion on the use of clinical practice guidelines (CPG) as defining the standard of care is warranted. Extensive reviews of the subject are available for the interested reader.[9,14-16] Several court cases have addressed the use of CPGs, and currently there is no set standard for how these documents are used in court cases. Some courts allow more liberal use of the CPGs, and others require more scrutiny as to the scientific validity of the CPG before it is admitted. Normally a document like a CPG would be considered "hearsay" in the courts , as the author is not available to testify or to allow for cross-examination. However, the court cases dealing with CPG use have suggested that if the guidelines are of some scientific validity, that they may be used as "learned treatises" and bypass the hearsay rule. CPGs may be used to lend credence to an expert witness, to impeach an expert witness, to defend a physician for following the document as the standard of care, or to suggest physician deviance from the document as deviance from the standard of care. In the end, an explanation by an expert as to why a CPG is indicative or not indicative of the standard of care goes a long way in a court case. When one side uses a CPG in a court case, it is up to the opposing side to ensure that the jury is given adequate

Table 2. Historical development of the Standard of Care

Initial Definition
Based on custom
That which is typically done is what is considered standard
The 20th Century Definition (*Helling v. Carey; The TJ Hooper*)
That which is customarily done plus anything that seems reasonable even if not typically done
The Modern Definition (*Hall v. Hilburn; McCourt v. Abernathy; Johnston v. St. Francis Medical Center*)
That which a minimally competent physician in the same field would do under similar circumstances

explanation as to why this may or may not actually represent the standard of care. This is a continually evolving topic and is currently dealt with on a case-by-case basis. Now recognizing the complicated issues that arise with formulation of CPGs, it would seem optimal that committees who develop these guidelines should allow flexibility, include multiple sources of scientific merit, and not be dependent on the opinion of a relatively small panel. In addition, if clear evidence is sparse, this should be openly acknowledged in the formation of the guideline.

SUMMMARY AND RECOMMENDATIONS

In conclusion, the concept of the standard of care has evolved over the years and will continue to change as legal theory in this area develops. Hopefully this will allow for increased certainty and clarity, which is the stated goal of all laws. The bad news is that there are several important cases where the suggestion is that even if a practice is not the standard, if it is reasonable, a physician can be found culpable for not pursuing that course of action. The good news for physicians is that in more recent cases the courts have frequently upheld that the standard of care is what a minimally competent physician in the same field would do in the same situation, with the same resources. These recent cases also note that bad outcomes are to be expected, and all entities can not be expected to be diagnosed. Finally, clinical practice guidelines are being used more frequently in court cases as support for the standard of care; however, their acceptance and uses are continually changing and decided on a case-by-case basis (Table 2).

Emergency physicians should be aware of these landmark cases that define the standard of care. In addition, physicians should be familiar with the content of various clinical practice guidelines so that one may practice by them, or document reason for deviating from them. Each state will also have statues that define malpractice in very specific terms. Physicians should review the relevant laws based on the state they practice in. By practicing with these concepts in mind, an emergency physician can feel more confident in daily practice, and when faced with a malpractice action. With this basic knowledge, the physician facing a suit may be able to assist his legal team in optimizing his/her defense.

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2010 *Western Journal of Emergency Medicine* Residency Abstract Competition

Thank you to all who submitted abstracts to the first annual "Resident Original Research Competition." We received submissions from 18 programs; all excellent examples of scholarly work done by the next generation of emergency medicine leaders. All authors are invited to submit a full manuscript for peer-review. *WestJEM* will publish the best of these papers.

The winner for 2010 – 2011 is an abstract submitted by Nicholas Caputo MD at Lincoln Medical and Mental Health Center entitled "Violent and Fatal Youth Trauma: Is There a Missed Opportunity?" With his co-investigators, Dr. Caputo documents the poor outcomes seen in patients caught up in the cycle of violence that many of our patients experience. Their research strengthens the argument that early intervention in this population has tremendous potential.

1. Violent and Fatal Youth Trauma: Is there a missed opportunity?

Nicholas D. Caputo, MD,MSc*, Christopher P. Shields, MD*, Cesar Ochoa, MD*, Jennifer Matarlo, RN†, Mark Leber, MD*, Robert Madlinger, DO†, Muhammed Waseem, MD*

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Objective: Accidents and assaults (homicides) are the leading causes of death among the youth of the United States. They account for 53.3% of deaths among children aged 1-19 years old. Youth violence is a significantly growing public health problem. Risk factors offered for the incidence of violent traumatic death include socioeconomic status, race, and place of residence (rural vs. urban). With five-year mortality rates for recidivism reach as high as 20%, it is important to determine whether victims with a history of violent trauma are at increased risk of fatal outcome. We hypothesize that victims of violent trauma who have one prior visit of violent trauma will have an increased odd of fatal outcome.

Methods: A retrospective chart review was conducted for patients presenting with penetrating trauma to the emergency department (ED) from January 1, 1999, to December 31, 2009. Any patient between the ages of 15 to 25 years old, who presented to the ED for any penetrating trauma were included. Patients with prior presentations for penetrating trauma were compared to those patients who were first time presenters to determine the odds ratio (OR), with statistical significance set at the $p<0.05$ level.

Results: Overall 15,395 patients were treated for traumatic presentations. Of these, 1044 patients met the inclusion criteria. Demographically, 79.4% were Hispanic, 19.4% were African-America and 0.96% was Caucasian. The average age was 21-years-old, and 98% of the population was male. One hundred and forty-seven (14%) had prior presentations, and 897 (86%) did not. Forty of the 147 patients (27%) with prior presentations had a fatal outcome compared to 29 patients of the 868 (3%), odds ratio 11.2 (95% CI 6.6-18.8, Pearson Chi Square $p<0.001$). A five-year mortality rate for those patients with fatal outcomes was calculated to be 16.5%.

Conclusion: Those who had prior ED visits for penetrating trauma were at greater risk of fatal outcomes compared to those with no prior visits. Therefore, trauma related ED visits might offer an opportunity for education and intervention. This may help to prevent future fatalities.

2. Vaginal Injuries After Consensual Versus Nonconsensual Sexual Intercourse In a Community-Based Population

Omar Kolonda, MD, Stanley Frye, MD, Paul Swiecicki MSIII, David Whalen, MD, Jeffrey S. Jones, MD

Grand Rapids Medical Education Partners/ Michigan State University Program in Emergency Medicine

Objectives: The spectrum of vaginal injuries from coitus can range from minor superficial trauma to life-threatening laceration or perforation. There is limited data involving the evaluation of patients with nonobstetric vaginal lacerations—almost all previous reports are case studies. The goal of this study was to describe the predisposing factors, injury patterns and treatment in women presenting with vaginal laceration due to consensual versus nonconsensual sexual intercourse.

Methods: Retrospective analysis of adult females with vaginal lacerations due to sexual intercourse in a cohort of patients who presented to the emergency departments at three urban United States academic medical centers, two rural community hospitals, as well as a sexual assault clinic. Over a four-year study period, all patients were examined by board-certified emergency physicians or forensic nurses, trained to perform medicolegal examinations using colposcopy with nuclear staining and digital photography. Demographics, assault characteristics and injury patterns were recorded using a standardized classification system. The main outcome variable was the frequency of severe vaginal lacerations (those requiring repair) in women presenting for treatment following consensual (CSI) versus nonconsensual sexual intercourse (NCSI).

Results: Four hundred twenty-seven cases met the inclusion criteria of the study; 65 (15%) reported CSI and 369 (85%) reported NCSI. Both groups were comparable in terms of age, ethnicity, time to physical examination, alcohol/drug use, marital status and prior sexual intercourse experience. NCSI subjects had a greater number of mean vaginal lacerations (1.7 vs. 1.1, $p<0.001$), smaller injuries (1.1cm vs. 4.1cm, $p<0.001$) which were generally located on the posterior vaginal wall (83% vs. 69%, $p=0.01$). NCSI vaginal lacerations were all superficial and did not require sutures. In contrast, 39% of CSI subjects (25/65) had lacerations sutured in the ED; three patients presented with hemorrhagic shock and required operative repair. Predisposing factors for vaginal lacerations were documented in 78% of CSI patients.

Conclusion: In this community-based population, severe vaginal lacerations were documented in women following consensual intercourse. A number of predisposing and etiologic factors may account for such injuries. Vaginal lacerations following sexual assault were more common but superficial in nature.

3. Does Chest CT Detect Clinically Significant Injuries Missed on Chest X-rays in Blunt Trauma Patients?

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Background: Although computed tomography (CT) has been shown to detect more injuries than plain radiography in blunt trauma patients, it is unclear whether these injuries truly change patient management.

Objectives: We sought to determine whether findings seen on chest CT [but not on chest radiograph (CXR)] result in significant changes in management.

Methods: The study was approved by human subjects committee. At an urban Level I trauma center, blunt trauma victims over 14-years-old who received emergency department chest imaging as part of their evaluation were enrolled. Significant intrathoracic injury (SITI) was defined as any of the following injuries on official radiology reports: pneumothorax, hemothorax, mediastinal hematoma, pneumomediastinum, sternal fracture, aortic injury, ruptured diaphragm, two or more rib fractures or pulmonary contusion. An expert panel, consisting of two emergency medicine attending physicians and an attending trauma surgeon, determined a priori the interventions that constituted significant change in patient management.

Results: Of the 1712 subjects were enrolled, 1435 (83.8%) patients had a CXR alone and 277 (16.2%) had CXR and CT. 10% (172) of all subjects were determined to have SITI: 20.9% on CXR alone, 28.5% on CT alone and 49.4% on both CXR and CT. In 17.7% of cases in which chest CT was performed, SITI was detected that was not seen on CXR: 28 multiple rib fractures, 16 pulmonary contusions, nine pneumothoraces, eight sternal fractures and one hemothorax. Four of these resulted in changes management: Two patients had chest tube placements and two patients were provided incentive spirometers for multiple rib fractures. Overall, chest CT led to a change in patient management in 1.4% (95% CI) of cases in which it was performed.

Conclusion: Although chest CT frequently detects injuries missed on CXR in blunt trauma patients, it rarely changes patient management.

HONORABLE MENTION FOR 2010-2011

Diagnostic Accuracy of Venous Blood Gas Electrolytes for the Diagnosis of Diabetic Ketoacidosis in the Emergency Department

Marc Probst, Michael Menchine, MD, MPH, Chad Agy C, Dianne Bach, Sanjay Arora, MD

University of Southern California, Los Angeles, CA

Predictors for Asthma Revisits in a Pediatric Emergency Department

William A. Stone Jr, MD, Muhammed Waseem, MD, Mark Leber, MD

Lincoln Medical and Mental Health Center, Bronx, NY

Motor Vehicle Collision in Relation to the Proximity of Electronic Billboard in a Large Urban Setting

Sam Cooper, MD, Craig L. Anderson, MPH, PhD, Camille Ferrer, Stacy Hata, Shahram Lotfipour MD, MPH, Bharath Chakravarthy, MD, MPH

University of California Irvine Medical Center, Orange, CA

The Effect of a Computerized Patient Care Report System on Physical Exam Documentation by a Collegiate BLS EMS Organization

Robert J. Katzer, MD, Samuel Adelman EMT, David J. Barton EMT, Sophie Clark EMT, Elizabeth L. Seaman EMT, Korin B. Hudson, MD, NREMT-P

Georgetown University/Washington Hospital Center

Correlating IVC Measurements with Intravascular Changes at Three Measurement Sites

Daniel Haase, MD, Shannon Gust, MD, Teresa Wu, MD, Kai-Ning Kohr, BS, MPH, David Drachman, PhD

Maricopa Medical Center, Phoenix, AZ

Do Emergency Physicians Order More Abdominal CT Scans when a Pediatric Surgery Service Is not Available?

Viraj S. Lakdawala, MD, Muhammed Waseem, MD

Lincoln Medical and Mental Health Center, Bronx, NY

FELLOWSHIP

in Emergency Medical Services and Disaster Medicine

University of California, Irvine School of Medicine

University of California, Irvine, Department of Emergency Medicine is seeking applicants for the fellowship in Emergency Medical Services and Disaster Medical Sciences for July 1, 2012. UCI Medical Center is a Level I Trauma center with 2,500 runs/year and a 37,000 ED census. Fellows serve as HS Clinical Instructors. The program combines the disciplines of emergency management/disaster medicine and public health with traditional EMS. A key focus of the fellowship is health policy and health services systems research including mass casualty management. Completion of American Council of Graduate Medical Education (ACGME) accredited emergency medicine residency required prior to start. A one or two-year program is available. The two-year program offers a master's degree, typically in Public Health. Salary is commensurate with level of clinical work. Send CV, statement of interest and three letters of recommendation to:

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

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EM FACULTY DEVELOPMENT FELLOWSHIP

UNIVERSITY OF CALIFORNIA, IRVINE SCHOOL OF MEDICINE

University of California, Irvine, Department of Emergency Medicine (EM) is seeking a HS Clinical Instructor- Faculty Development Fellow for July, 2012. University of California, Irvine Medical Center is a Level I Trauma center with 2,500 runs/year, 37,000 ED census with a three-year residency program since 1989. Fellowship concentration areas include residency education, medical school curriculum design, journal editing and publishing. This two-year faculty development fellowship includes a requirement for completion of a Masters in Education or Public Health. Possibility exists for a one-year fellowship for candidates with a Masters degree or those starting one during their fellowship. Completion of an ACGME accredited EM Residency is required. Salary is commensurate with the level of clinical work.

Send CV and statement of interest to Fellowship co-Directors:
Dr. Shahram Lotfipour at SHL@uci.edu
Dr. Bharath Chakravarthy at bchakrav@uci.edu
or
Department of Emergency Medicine
UC Irvine Medical Center, Route 128-01
101 The City Drive
Orange, CA 92868
or
Call 714-456-5239

See the Department of Emergency Medicine's website available at http://www.emergencymed.uci.edu/fellowships.asp for more details.

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Dfarcy@me.com
David A. Farcy, MD, FAAEM
President FLAAEM.

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Ultrasound Fellowship, University of California, Irvine,
Department of Emergency Medicine

Seeking a Health Sciences Clinical Instructor (Fellow in Emergency Ultrasound) as an ongoing recruitment. University of California, Irvine is a Level I Trauma, cardiac, stroke and burn receiving center with 2,500 trauma activations/year and a 37,000 ED patient census. Division of Emergency Ultrasound established 2001. US Director is fellowship trained and RDMS certified, along with five other faculty members. We operate a robust emergency Ultrasound rotation for internal and external medical students and residents, with an active research, teaching and clinical environment. Clinical schedule of eight 10-hour shifts per month. Salary commensurate with level of clinical work. Fellow will perform 1200 scans/year with tape review of 4000 more. RDMS certification expected upon completion of the fellowship. Completion of EM residency required prior to starting fellowship. Please send CV, statement of interest and three letters of recommendation to: J Christian Fox, MD RDMS at jfox@uci.edu Department of Emergency Medicine, Route 128, UC Irvine Medical Center, 101 City Drive South, Orange, CA 92868.

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Pan-Pacific Emergency Medicine Congress 2012

PEMC
2012

Hand-in-Hand Across the Pacific

October 23-26, 2012
Coex, Seoul, Korea

For the first time, the American Academy of Emergency Medicine (AAEM) and the Korean Society of Emergency Medicine (KSEM) organize the Pan-Pacific Emergency Medicine Congress 2012 (PEMC 2012), in Seoul, Korea, on October 23-26, 2012. The latest issues and various areas that cover the emergency medicine will be discussed.

Approximately 1,000 attendees will join the congress. Renowned scholars and experts will offer sessions and workshops in areas of interest to those involved in emergency medicine around the world.

PEMC 2012 will provide every participant with an excellent opportunity to exchange ideas, build new networks, strengthen old ones, and keep up with what is happening in the emergency medicine.

Mark your calendar for the PEMC 2012.
For more information, visit at
www.pemc2012.org.

Hosted by

The Korean Society of Emergency Medicine

American Academy of Emergency Medicine

40th Annual

CAL/ACEP Scientific Assembly

& Ultrasound Workshop

June 23-25, 2011 - Newport Beach Marriott

Newport Beach, California

This conference is sponsored by The American College of Emergency Physicians and CAL/ACEP.

Thursday, June 23

Anaphylaxis: Should the Recent Guidelines Change Our Practice? (1 hour) *Scott Votey, MD*
Understand the pathophysiology of anaphylaxis and how it influences treatment choices; Become aware of the range of presentations of anaphylaxis so as to be able to promptly diagnose patients presenting atypically; Develop a severity-based pharmacologic therapy regimen for anaphylaxis; Become aware of the current standards in the management of anaphylaxis including the appropriate use of epinephrine.

Glands Gone Wild: Endocrine Emergencies (1 hour) *William Mallon, MD, FACEP*
Endocrine emergencies represent a group of potentially life-threatening conditions that are frequently overlooked, resulting in delays in both diagnosis and treatment. Billy will highlight factors that contribute to their high mortality rates.

Difficult Dislocations (1 hour) *Greg Hendey, MD, FACEP*
To demonstrate innovative techniques for the reduction of difficult dislocations, using multiple video clips; To assess the risk of neurovascular compromise after a joint dislocation and plan a reasonable evaluation; To discuss sedation and anesthesia options for facilitating reduction techniques.

Recent EM Literature that Will Change Your Practice (1 hour) *Sanjay Arora, MD*
A review of the most significant studies published throughout the medical literature in past years. Each article presented will be assessed to determine its relevance to the practice of clinical emergency medicine. This lecture will identify advances in emergency medicine by reviewing the recent literature, describe the limitations of recent studies on the practice of emergency medicine, and discuss the implications of recent studies regarding clinical emergency medicine.

RESEARCH FORUM (3 hours) *Matthew Lewin, MD, PhD, FACEP*
Find out what's on the cutting edge of research from colleagues around the state. Ten abstracts will be presented and Awards for Best Research, Best Presentation and Most innovative Project will be given. Upon completion of this course, participants will be able to discuss the pros and cons of the results of a moderated oral research abstract, identify research/treatment that could be applied to clinical practice, and explain research trends occurring in emergency medicine.

ULTRASOUND IV WORKSHOP (3 hours) *Laleh Gharahbaghian MD, FAAEM, Martine Sargent, MD David Francis, MD, and Brita Zaia, MD*
This ultrasound technique is safe, rapid, portable, and noninvasive; it allows rapid bedside diagnosis and increases success and speed for many procedures. This introduction course will cover lectures on ultrasound basics, literature, anatomy, technique, and pitfalls and complications, as well as a hands-on lab portion. Register today, while space is still available.

| *Meetings* | ***CEMAF**, CHA. EMREF, EMRA, CalEMRA Residents **Conference*** | *Events* | *Opening Reception, Awards Luncheon, Presidents Dinner*

Friday, June 24

Dealing with Difficult Parent (1 hour) *Ghazala Sharieff, MD, FACEP*
To be familiar with the impact of antibiotic use on the development of asthma, and diarrhea; To know the clinical guidelines for obtaining a head CT in children with head injury; To know the AAP guidelines on teenage drug testing.

Electrolyte Emergencies (1 hour) *Vena Ricketts, MD, FACEP*
Recognize the clinical presentations of patients presenting to the ED with Electrolyte Emergencies; Participants will have a distinctive concise knowledge on the management of Electrolyte Emergencies; Participants will learn several clinical pearls on evaluation and management; Learn how to avoid potential disasters.

LLSA Review (3 hours) *Peter D'Souza, MD, FACEP*
Review key articles of Emergency Medicine literature as determined by the American Board of Emergency Medicine; Review key questions from the readings as determined by the American Board of Emergency Medicine.

Saturday, June 25

Therapeutic Hypothermia Post-Cardiac Arrest: Evidence to Practice (1 hour) *H. Bryant Nguyen, MD*
To review guidelines for post-cardiac arrest care; To review the evidence for therapeutic hypothermia/ targeted temperature management post-cardiac arrest; To discuss cooling methods; To discuss best practices and implementation issues for a post-cardiac arrest care bundle.

Pediatric Literature Review (1 hour) *Maureen McCollough, MD, FACEP*
Bedside ultrasound has dramatically changed the practice of emergency medicine for adult patients and is just beginning to change the face of pediatric emergency medicine. This course will be a great review of the most recent articles covering a wide variety of pediatric emergency medicine topics from fever and respiratory illness to abdominal pain and trauma, this lecture will definitely change your practice.

Cool Tox Tricks: Simple Solutions for Poisoned Patients (1 hour) *Rais B. Vohra, MD*
This lecture will cover 6 clinical cases in poison management with 6 simple solutions for busy ER doctors that are easy to learn, efficiency-boosting, and evidence-based.

Reversal of Anticoagulation in Life Threatening Bleeding (1 hour) *Mark I. Langdorf, MD, FACEP*
Learn the indications and contraindications to reversal of anticoagulation in patients with intracranial hemorrhage; Appreciate the controversies in management; Understand the limited research in this area; Learn reversal strategies for Coumadin, Heparin, aspirin and Plavix.

Faculty

Fred Abrahamian, DO, FACEP
Scientific Assembly Program Chair
Assistant Professor of Medicine/Emergency Medicine
UCLA School of Medicine
Director of Education, Department of Emergency Medicine, Olive View-UCLA Medical Center

Matthew Strehlow, MD
CAL/ACEP Education Committee Chair
Clinical Assistant Professor of Surgery/Emergency Medicine, Associate Medical Director
Director, Clinical Decision Area
Stanford University Emergency Department,
Division of Emergency Medicine, Stanford, California

Sanjay Arora, MD
Associate Professor of Clinical Emergency Medicine, University of Southern California, Keck School of Medicine, Los Angeles County Hospital

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Clinical Instructor of Surgery, Division of Emergency Medicine, Stanford University School of Medicine

Dave Francis, MD
Fellow Emergency Ultrasound, Clinical Instructor of Surgery, Division of Emergency Medicine, Stanford University Hospital & Clinics Emergency Medicine

Laleh Gharahbaghian MD, FAAEM
Associate Director, Emergency Ultrasound; Co-Director, Emergency Ultrasound Fellowship, Stanford University Medical Center, Division of Emergency Medicine Department of Surgery, Stanford, California

Greg Hendey, MD, FACEP
Clinical Professor, Department of Emergency Medicine, University of Michigan Medical School; Staff Physician, St. Joseph Mercy hospital System; Staff Physician, Chelsea Hospital, Chelsea, Michigan; CEO, Medical Practice Risk Assessment, Inc; Risk Management Consultant, Emergency Physicians Medical Group, Ann Arbor, Michigan; President, Savannah Assurance, Limited, Bridgetown, Barbados; Past President, ACEP

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Martine Sargent, MD
Ultrasound Director, Assistant Professor, UCSF Department of Emergency Medicine San Francisco General Hospital & Trauma Center

Ghazala Sharieff, MD, FACEP
Division Director, Emergency Department, Rady Children's hospital and health Center/ Clinical Professor, University of California, San Diego; Director, Pediatric Emergency Medicine, Palomar-Pomerado Hospital/ California Emergency Physicians, San Diego, California

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Program Director, UCLA/Olive View-UCLA Emergency Medicine Residency Program

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Attending Physician and Clinical Instructor, Department of Emergency Medicine, Kaiser Permanente Medical Center, San Francisco, California

FACULTY: Those involved in the planning and teaching of this activity are required to disclose to the audience any relevant financial interest or other relationship. All faculty, planners, and staff in a position to control the content of this CME activity have indicated that he/she has no relationship, which could be perceived as a potential conflict of interest.

Accreditation

Physicians: This activity has been planned and implemented in accordance with the Essential Areas and Policies for the Accreditation Council of Continuing Medical Education through joint sponsorship of ACEP and CAL ACEP. The American College of Emergency Physicians is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. The American College of Emergency Physicians designates this live activity for a maximum of 17.00 *AMA PRA Category 1 Credit(s)*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.
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EMTs/Paramedics: EMREF is approved by the Sacramento County EMS Agency for 17 Continuing Education Units, Provider Number 34-4600.
DO's: American Osteopathic Association (AOA) recognizes ACCME Category 1 Credit as AOA Category 2-A Credit. All members of AOA are required to participate in CME programs to meet the *2010-12 CME Cycle* of 15 Credits ACCME Category 1 Credits and 15 AOA Category 1-A Credits.

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June 23 – 25, 2011

Ultrasound Workshop

June 23 & 24, 2011

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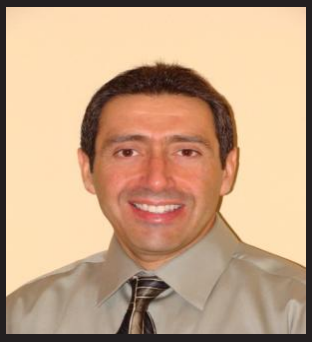

Program Chair: Fredrick M. Abrahamian, D.O.