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THE AMERICAN JOURNAL OF EMERGENCY MEDICINE (ISSN 0735-6757) is published seven times a year (January, March, May, July, September, October, November) (For Post Office use only: Volume 23 issue 2 of 7) by Elsevier, 170 S. Independence Mall West, Suite 300E, Philadelphia, PA 19106-3399. Periodicals postage paid at Orlando, FL 32887-4800 and additional mailing offices.

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Original Contributions

Severe upper extremity injuries in frontal automobile crashes: the effects of depowered airbags

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Received 5 February 2004; accepted 5 February 2004

Abstract

Background: The purpose of this study was to determine the effects of depowered frontal airbags on the incidence of severe upper extremity injuries.

Methods: The National Automotive Sampling System database files from 1993 to 2000 were examined in a study that included 2,413,347 occupants who were exposed to an airbag deployment in the United States.

Results: Occupants exposed to a depowered airbag deployment were significantly more likely to sustain a severe upper extremity injury (3.9%) than those occupants exposed to a full-powered airbag deployment (2.5%) ($P = .01$). Full-powered systems resulted in an injury distribution of 89.2% fractures and 7.9% dislocations compared with depowered systems with 55.3% fractures and 44.3% dislocations.

Conclusions: Although depowered airbags were designed to reduce the risk of injuries, they appear to have increased the overall incidence of severe upper extremity injuries through a shift from long bone fractures to joint dislocations.

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1. Introduction

Although airbags have reduced the incidence of fatal and severe injuries in automobile collisions, they have been shown to increase the risk of other injuries [1]. These associated minor injuries include corneal abrasions, skin contusions or lacerations, and upper extremity injuries [2-20]. In particular, upper extremity injuries have been identified through case reports that present a wide range of upper extremity injuries, from a minor abrasion to a more severe avulsion or fracture [21-35]. Although upper extremity injuries were observed before airbag implementation, it is suggested that the risk of serious upper extremity injury to restrained occupants with airbags is higher when compared with those without airbags [22,36,37]. Upper extremity injuries have been estimated as nearly a quarter of all injuries to the whole body in motor vehicle crashes [33,38].

To investigate the interaction between the upper extremity and a deploying frontal airbag, experimental testing has been done using Hybrid III and Research Arm Injury Device upper limbs [36,39-41]. In addition, numerous cadaver studies have aimed to estimate dynamic injury tolerance and to reproduce fractures similar to those observed in real-world case studies [42-46]. As identified in case studies, injuries to the upper extremity can occur because of contact with the airbag during or after deployment, and are likely caused by various combinations of axial and bending moments applied to the arm [25].
To reduce the incidence of airbag-induced fatal and severe injuries to small females and children, the National Highway Traffic Safety Association submitted a change in safety standards [37]. The new safety regulations, effective in 1998 model vehicles, allowed automobile manufacturers to reduce the power of the deploying airbag and still meet the safety standards by passing a standardized sled test rather than a full-vehicle crash test. The new depowered airbags could be less aggressive than the pre-1998 full-powered airbags.

Several recent studies have shown that depowered airbags did reduce the risk of serious injuries as well as changing the overall injury patterns [47-50]. Fewer injuries occurred to drivers of the later models vehicles. The depowered airbags could be responsible for these improvements. Although previous studies have provided insight into the interaction between an airbag and the upper extremity, the national rate of incidence of severe upper extremity injuries is unknown for occupants exposed to full-powered and depowered airbags. The purpose of this paper is to determine the effects of depowered airbags on the overall risk and severity of upper extremity injuries in frontal automobile crashes.

2. Methods

This study uses the National Automotive Sampling System (NASS) to eliminate the inaccuracies associated with small case study projections [51]. The primary advantage of using the NASS is that the database includes an analysis of approximately 5000 cases per year and it allows for national incidence estimates. The injuries are coded by trained nurses using the Abbreviated Injury Scale (AIS) [52]. This coding allows for a consistent and accurate distinction and identification of upper extremity injuries. The NASS database has been used for national injury projection studies to analyze injury severity and crash characteristics for things such as lower extremity injuries and restraint effectiveness in motor vehicle crashes [1,5,53-59]. Every crash investigated for the NASS database is assigned a weighted value, which scales the incidence of the particular crash investigated to a number that represents actual occurrence of similar non-investigated crashes that occur in the United States each year. Unweighted numbers reflect actual values counted from the cases that appear in the NASS database. The AIS scale classifies injuries by body region on a 6-point scale ranging from low severity (AIS1) to fatal (AIS6). The AIS values are assigned for each injury sustained and do not include combined effects from multiple injuries to the same patient.

For this study, cases in the NASS with an airbag deployment were selected from an 8-year span, years 1993 through 2000, that included drivers and front seat occupants only, and excluded ejected occupants and rollovers. In addition, only frontal impacts were considered, which are defined as having a primary direction of force of 11, 12, or 1 o’clock. Only severe upper extremity injuries were analyzed, identified as AIS level 2 severity and higher. Such injuries include amputation, avulsion, burn, crush, dislocation, fracture, and laceration. The upper extremity was defined to include the acromium, clavicle, scapula, humerus, elbow, radius, ulna, wrist, hand, and fingers. The injuries and specific body region were identified in the NASS database using the current AIS injury codes. Injuries to the fingers, hand, and wrist were all grouped together and termed hand injuries. Frequencies of occupants with injuries and total injuries to occupants were analyzed. This study is divided into 3 parts.

2.1. Depowered and full-powered airbag deployment

For all occupants who were exposed to a full-powered airbag deployment, the number of occupants that sustained a severe upper extremity injury was compared with the total number of occupants who did not sustain a severe upper extremity injury. Next, an analogous search was performed for crashes with a depowered airbag deployment. For all occupants who were exposed to a depowered airbag deployment, the number of occupants that sustained a severe upper extremity injury was compared with the total number of occupants who did not sustain a severe upper extremity injury. Frequencies of occupants and injuries were analyzed. The top 3 sources of injury were identified for occupants exposed to full-powered and depowered airbag deployments.

2.2. Severe upper extremity injury types and locations

Severe upper extremity injuries were further examined to compare injury types and locations, depending on which type of airbag the occupant was exposed to and whether or not the airbag was the source of the injury. Specific severe upper extremity injury types were compared as percentages of total upper extremity injuries in similar crashes, depending on the injury source. To further analyze injuries by location, upper extremity fractures were broken down by specific body region to compare resulting fracture location by airbag type and injury source.

2.3. Occupant and crash characteristics

Various occupant and crash characteristics were examined to identify trends that correlate with incidence of severe upper extremity injury for occupants exposed to airbag deployment. The first study involved a comparison between the types of occupants exposed to each type of airbag deployment. This part identified differences between the population exposed to full-powered airbags and those exposed to depowered airbags. Group 1 was the group of
occupants exposed to a full-powered airbag deployment, whereas group 2 was the group of occupants exposed to a depowered airbag deployment. Average values and standard deviations were calculated for occupant height, weight, age, sex, seat position, seatbelt use, and change in velocity ($\Delta V$). Next, a similar investigation was performed for occupants with airbag-induced severe upper extremity injuries—depending on whether the airbag was full-powered or depowered. Group 1-A was the group with an airbag-induced injury from a full-powered airbag, whereas group 2-A was the group of occupants with an airbag-induced injury from a depowered airbag. Average values and standard deviations were calculated for occupant height, weight, age, sex, seat position, seatbelt use, and $\Delta V$.

3. Results

3.1. Depowered and full-powered airbag deployment

A total of 2,413,347 occupants from 6,091 cases were exposed to an airbag deployment between the years 1993 and 2000. Because the proportion of airbag-equipped vehicles in the fleet is increasing, more occupants are exposed to airbag deployments each year. Accordingly, the number of occupants who sustained a severe upper extremity injury in a crash with airbag deployment has also increased. In addition, every year there have been more occupants who sustained a severe upper extremity injury when exposed to a full-powered airbag deployment than when exposed to a depowered airbag deployment (Fig. 1).

Occupants were significantly more likely to sustain a severe upper extremity injury when exposed to a depowered airbag than when exposed to a full-powered airbag ($P = .01$) (Fig. 2). In particular, 2.5% of occupants exposed to a full-powered airbag deployment sustained a severe upper extremity injury compared with 3.9% of occupants exposed to a depowered airbag. In addition, 0.7% of occupants who were exposed to a full-powered airbag deployment sustained a severe upper extremity injury specifically from the airbag compared with 0.8% of those occupants exposed to a depowered airbag ($P = .67$).

There were 88,324 total severe upper extremity injuries to occupants, 68,691 of which were to occupants exposed to a full-powered airbag deployment (77.8%), whereas 19,633 occurred to occupants who were exposed to a depowered airbag deployment (22.2%) (Fig. 3). The top 3 injury sources for occupants who sustained a severe upper extremity injury when exposed to a full-powered airbag deployment were the airbag (30.4%), the steering wheel (17.9%), and the instrument panel or glove box (14.0%). If the occupants were exposed to a depowered airbag deployment.
deployment, the leading source for the injuries was the instrument panel or glove box (41.8%), followed by the airbag (18.3%) and the steering wheel (17.3%).

### 3.2. Severe upper extremity injury types and locations

Severe upper extremity injuries rated as AIS2 and AIS3 were grouped together for this analysis. For occupants who sustained severe upper extremity injuries from sources other than the airbag, the majority were fractures for both occupants exposed to a full-powered airbag deployment (87.5%) and occupants exposed to a depowered airbag deployment (96.5%) (Table 1). However, there was a shift in severe upper extremity type for injuries that were induced specifically by the airbag. For occupants with airbag-induced injuries from a full-powered airbag, 89.2% were fractures, whereas occupants who sustained airbag-induced injuries from a depowered airbag sustained only 55.3% as fractures, whereas 44.3% were dislocations. Of these, 91.7% were shoulder dislocations, whereas 8.3% were dislocations of the wrist or fingers. There were no airbag-induced elbow dislocations from a depowered airbag.

There was a shift in the fracture location depending on whether the occupant was exposed to a full-powered or depowered airbag deployment, and whether the airbag was the source of the fracture (Table 2). For injuries that were airbag induced, occupants exposed to a full-powered airbag sustained the majority to the radius (54.2%), followed by the ulna (35.7%). There were no airbag-induced fractures to the scapula from a full-powered airbag. In contrast, occupants who were exposed to a depowered airbag sustained most of the airbag-induced injuries to the humerus (30.6%), and the radius (30.5), followed by the scapula (19.9%). There were no airbag-induced fractures to the ulna from a depowered airbag.

### 3.3. Occupant and crash characteristics

The next analysis was made by examining occupant and crash characteristics for the 2,413,347 occupants who were exposed to an airbag deployment. There was no significant difference in the continuous variables between those

### Table 1  Comparison of severe (AIS2 and AIS3) upper extremity injury types for occupants exposed to a full-powered or depowered airbag deployment

<table>
<thead>
<tr>
<th>Source: airbag</th>
<th>Source: other</th>
<th>Source: airbag</th>
<th>Source: other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full-powered airbag deployment</td>
<td>Depowered airbag deployment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amputation</td>
<td>0</td>
<td>675</td>
<td>0</td>
</tr>
<tr>
<td>Avulsion</td>
<td>19</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Burn</td>
<td>499</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Crush</td>
<td>0</td>
<td>796</td>
<td>0</td>
</tr>
<tr>
<td>Dislocation</td>
<td>1,645</td>
<td>3,146</td>
<td>1,594</td>
</tr>
<tr>
<td>Fracture</td>
<td>18,600</td>
<td>41,858</td>
<td>1,993</td>
</tr>
<tr>
<td>Laceration</td>
<td>90</td>
<td>879</td>
<td>0</td>
</tr>
<tr>
<td>Unknown</td>
<td>0</td>
<td>468</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td>20,853</td>
<td>47,838</td>
<td>3,602</td>
</tr>
</tbody>
</table>

NFS indicates not further specified.

### Table 2  Comparison of severe (AIS2 and AIS3) upper extremity fracture locations for occupants exposed to a full-powered or depowered airbag deployment

<table>
<thead>
<tr>
<th>Source: airbag</th>
<th>Source: other</th>
<th>Source: airbag</th>
<th>Source: other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full-powered airbag deployment</td>
<td>Depowered airbag deployment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acromion</td>
<td>0</td>
<td>26</td>
<td>0</td>
</tr>
<tr>
<td>Arm NFS</td>
<td>90</td>
<td>1,719</td>
<td>28</td>
</tr>
<tr>
<td>Clavicle</td>
<td>206</td>
<td>6,667</td>
<td>5</td>
</tr>
<tr>
<td>Forearm NFS</td>
<td>10</td>
<td>0</td>
<td>147</td>
</tr>
<tr>
<td>Hand</td>
<td>1,238</td>
<td>11,233</td>
<td>200</td>
</tr>
<tr>
<td>Humerus</td>
<td>332</td>
<td>3,625</td>
<td>609</td>
</tr>
<tr>
<td>Radius</td>
<td>10,089</td>
<td>9,010</td>
<td>607</td>
</tr>
<tr>
<td>Scapula</td>
<td>0</td>
<td>171</td>
<td>397</td>
</tr>
<tr>
<td>Ulna</td>
<td>6,635</td>
<td>9,407</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td>18,600</td>
<td>41,858</td>
<td>1,993</td>
</tr>
</tbody>
</table>

NFS indicates not further specified.
occupants exposed to a full-powered or depowered airbag deployment (Table 3). However, drivers were significantly more likely to be exposed to a full-powered airbag than were passengers (P = .01). In particular, 88.1% of drivers exposed to an airbag were exposed to a full-powered airbag deployment, compared with 81.8% of passengers exposed to airbags. In addition, of all occupants exposed to a full-powered airbag, 85.5% were drivers, compared with 78.1% drivers for those occupants exposed to a depowered airbag.

Females were 49.1% of the population exposed to full-powered airbags, compared with 51.5% of the occupants exposed to depowered airbag deployments. In addition, for all occupants exposed to an airbag deployment, 86.7% of females were exposed to full-powered airbags, compared with 87.7% of males who were exposed to airbags. This difference was not found to be significant (P = .67). Finally, there was no difference in the use of seatbelts for occupants exposed to depowered or full-powered airbags (P = .87). In particular, for all occupants exposed to airbags, 87.8% of unbelted occupants and 87.2% of belted occupants were exposed to full-powered airbags. In addition, 85.4% of occupants were belted when exposed to a full-powered airbag, whereas 86.1% were belted when exposed to a depowered airbag deployment.

The next analysis involved a comparison between the occupants that sustained a severe airbag-induced upper extremity injury when exposed to a full-powered or depowered airbag (Table 4). Within a 95% confidence interval, occupant height, weight, age, and crash ΔV were all not significant factors correlating with incidence of airbag-induced injury based on airbag type.

There was no significant difference in incidence of airbag-induced severe upper extremity injury for occupants exposed to depowered or full-powered airbag deployment based on the occupant seat position (P = .66), sex (P = .13), or use of seatbelts (P = .78). In particular, 85.9% of drivers with airbag-induced severe upper extremity injuries were exposed to a full-powered airbag deployment, compared with 81.1% of passengers with airbag-induced severe upper extremity injuries. In addition, of all occupants with airbag-induced severe upper extremity injuries from full-powered airbags, 65.9% were drivers, compared with 57.6% of those occupants with airbag-induced severe upper extremity injuries from depowered airbags. Of all females with airbag-induced severe upper extremity injuries, 93.5% were exposed to full-powered airbags, compared with 64.5% of males. In addition, of all the occupants with airbag-induced severe upper extremity injuries from full-powered airbags, 75.5% were female, compared with 28.1% of occupants with airbag-induced severe upper extremity injuries from depowered airbags. In addition, there was no difference in the use of seatbelts for occupants with airbag-induced severe upper extremity injuries from depowered or full-powered airbags. In particular, 88.1% of unbelted occupants with airbag-induced severe upper extremity injuries were exposed to full-powered airbags, compared with 83.5% of belted occupants. Finally, of those occupants with airbag-induced severe upper extremity injuries from full-powered airbags, 85.9% were belted, compared with 89.9% of occupants with airbag-induced severe upper extremity injuries from depowered airbag deployment.

4. Discussion

This paper presents the most comprehensive upper extremity injury study to date concerning the comparison between depowered and full-powered airbags. It investigates 25,464 individual cases over 8 years to identify the effects of depowered frontal airbags on the incidence of upper extremity injuries for occupants exposed to a frontal airbag deployment.

In contrast to previous experimental research with human cadaver arms, this study found the risk of severe injury increases from 2.5% to 3.9% with exposure to depowered airbags [36,42,44,45]. This is likely because the previous research focused on radius and ulna fracture prediction as the risk of joint dislocation was unknown. It is suggested that future experimental studies be performed to investigate the injury biomechanics of upper extremity joint dislocations. These data would be useful for designing future airbags to reduce the risk of both fractures and dislocations.
Acknowledgment

The authors thank JP Research for their assistance with the case selection and statistical analysis.

References

[52] Association for the Advancement of Automotive Medicine (AAAM). The Abbreviated Injury Scale (AIS); 1998. [Revision, Des Plains, Ill].
Lack of relationship between hypertension-associated symptoms and blood pressure in hypertensive ED patients

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Received 16 December 2003; accepted 16 February 2004

Abstract A number of cardiopulmonary and neurological symptoms are presumed to be associated with hypertension. We examined the prevalence of these symptoms in ED patients with elevated blood pressure (BP) and studied the relationship between symptom prevalence and BP value. We enrolled consecutive adult ED patients with sustained BP elevation (systolic BP ≥140 mm Hg, diastolic BP ≥90 mm Hg). BP values were categorized according to Joint National Committee on Prevention, Evaluation, and Treatment of High Blood Pressure, 6th Report criteria. Elevated BP was noted in 551 (29%) of 1908 patients. Unprompted complaints of hypertension-associated symptoms were noted in 26%, and there was no association between BP category and complaints other than dyspnea. Symptom interviews were conducted in 294 (56%) patients; 68% of this subset noted ≥1 current hypertension-associated symptom with no relationship between symptom prevalence and BP category. We conclude that symptoms putatively associated with hypertension are common among ED patients with elevated BP, and their prevalence appears unrelated to BP value.

1. Introduction

A number of cardiovascular, pulmonary, and neurological symptoms are believed to be associated with hypertension. Headache, visual changes, chest pain, dyspnea, and focal neurological deficits are widely considered potential symptoms of blood pressure (BP) elevation and suggestive of acute hypertensive end-organ dysfunction [1-5]. Although not considered a sign of end-organ damage, epistaxis is also considered a hypertension-related symptom. The presence of any of these complaints in a patient with elevated BP may lead physicians to perform an extensive evaluation to exclude hypertensive emergency, defined as a situation requiring immediate BP reduction to mitigate acute end-organ dysfunction [2]. Some authorities regard the presence of these symptoms in a patient with significant BP elevation to be diagnostic of hypertensive emergency [1,6,7].

Many of the symptoms putatively associated with hypertension are common among patients presenting to the ED [8]. Although prior investigations have not detected a strong relationship between symptom prevalence and BP values among chronically hypertensive patients, these were studies of stable patient populations, and the findings may...
Hypertensive symptoms

not be applicable to the ED population [9,10]. To our knowledge, there are no systematic studies in the ED or other acute care setting of the association between the degree of BP elevation and the presence of symptoms suggestive of hypertension-related end-organ damage.

We undertook a study of the association between the presence of hypertensive symptoms and BP values among ED patients with elevated BP. In part 1 of the study, chief complaints of all ED patients with elevated BP were extracted from the medical record. In part 2, patients were interviewed regarding current and recent symptoms. Our null hypothesis was that hypertension-associated symptoms, either as recorded in the medical record or elicited in interviews, are not more frequent among patients with greater degrees of BP elevation.

2. Methods

2.1. Study design

This was a prospective observational investigation. The protocol was approved by the medical school’s institutional review board. No change in medical management was required for this study. On-duty physicians, nurses, and students were not informed of the purpose of the investigation.

2.2. Study setting and population

The study was performed in the ED of an urban teaching hospital with approximately 50,000 annual adult visits and serving a predominantly African-American population. The ED is staffed by attending physicians board-certified or board-eligible in emergency medicine, by emergency medicine residents, and by residents in other specialties performing emergency medicine rotations under the supervision of the emergency medicine attending physicians.

2.3. Study protocol

Consecutive patients at least 18 years presenting to the ED during a 14-day enrollment period were eligible for participation. All patients had their BP measured by ED nurses using an automated BP device, calibrated to the manufacturer’s specifications (Welsh-Allyn Propaq Encore, Beaverton, Ore). The nurses had been trained by the manufacturer in the use of the device and were instructed to use a cuff of appropriate size. Patients arriving via triage had their BP measured in a seated position; those arriving by ambulance or taken directly to the treatment area had their BP measured while seated or supine on a stretcher. Patients with initial systolic BP values ≥140 mm Hg or initial diastolic BP values ≥90 mm Hg had their BP repeated at least 10 minutes later. Patients were included in the study if their systolic BP remained ≥140 mm Hg or diastolic BP remained ≥90 mm Hg upon repeat measurement. The only exclusion to enrollment was prior participation in the study.

Research associates were present in the ED at all times during the study period and were trained in data collection for this investigation. The associates reviewed all BP values recorded in triage and throughout the patients’ ED stay to determine study eligibility. Demographic information and all BP values obtained in the ED were recorded by the research associates on a standardized data collection instrument.

In part 1 of the study, the chief complaint of study patients was extracted from the medical record by the research associates. The primary source of this information was the triage nurses’ notes. For patients bypassing triage, the chief complaint was abstracted from the physician ED record. All patients meeting study enrollment criteria were included in part 1.

In the second part of the study, research associates approached all alert and oriented subjects for consent to undergo a detailed structured interview. Patients providing consent were questioned regarding the presence of symptoms typically cited as associated with hypertension, specifically, shortness of breath, chest pain, dizziness, headache, weakness, or numbness on one side of the body, nosebleed, and change in vision [1,6,7]. These symptoms are referred to hereafter as “hypertension-associated symptoms.” Patients were asked whether each symptom was present at the time of ED arrival, in the past 24 hours, or in the week before ED presentation. Positive responses were not mutually exclusive.

2.4. Data analysis

Criteria established by the Joint National Committee on Prevention, Evaluation, and Treatment of High Blood Pressure, 6th Report (JNC-VI), were used to classify patients as having stage 1, stage 2, or stage 3 BP values, as presented in Table 1 [1]. When systolic and diastolic values were in different stages, patients were classified according to the greater stage. The greatest BP value obtained during the ED visit was used as the reference value in assigning BP stages for analysis.

Intergroup differences in the prevalence of each hypertension-associated symptom and cumulative symptom prevalence were assessed using the \( \chi^2 \) statistic or Fisher exact test, as appropriate, using a significance threshold of \( P < .05 \). Cochran’s linear trend statistic was used to assess intergroup trends in symptom prevalence. Independent assessments were planned of the data obtained in part 1, in which complaints were determined from the medical

<table>
<thead>
<tr>
<th>Table 1 BP staging criteria</th>
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</thead>
<tbody>
<tr>
<td>Category</td>
</tr>
<tr>
<td>Not hypertensive</td>
</tr>
<tr>
<td>Stage 1</td>
</tr>
<tr>
<td>Stage 2</td>
</tr>
<tr>
<td>Stage 3</td>
</tr>
</tbody>
</table>

When systolic and diastolic BP values fall into different categories, the higher category is used to classify the subject. Adapted from the JNC-VI report [2].
record of all eligible patients, and the data obtained in part 2, in which symptoms were elicited from consenting patients.

3. Results

During 14 days of continuous data collection, 1908 patients presented to the ED. Elevated BP was noted in 551 (29%) patients. Twenty-two (4%) patients with elevated BP were excluded from further analysis because of incomplete data collection; none of these patients had consented to interview. Of the remaining 529 analyzable patients, 294 (56%) provided consent for interview, 200 (38%) were not approached for an interview due to severity of illness or inability to provide consent, and 35 (6%) declined to be interviewed.

The median subject age was 51 years (interquartile range [IQR], 39 to 65 years). Fifty-four percent of subjects were women. The majority of subjects (412 [78%]) were African American, with 43 (8%) Latino, 42 (8%) white (non-Latino), and 32 (6%) Asian subjects. The median systolic BP was 152 mm Hg (IQR, 142 to 170), and median diastolic BP was 88 mm Hg (IQR, 80 to 96). BP values were categorized as stage 1 in 304 subjects (57%), stage 2 in 127 (24%), and stage 3 in 98 (19%). No differences in sex, ethnicity, or BP values were noted between patients who consented for interview and those who did not. No differences were noted between excluded patients and those included in the final analysis.

Part 1 of the study assessed the prevalence of symptoms recorded in the medical record. As shown in Table 2, 26% of study patients had chief complaints of one or more hypertension-associated symptoms. Chest pain and shortness of breath were the most frequent symptoms. Dyspnea was found to be significantly more common among patients with stage 3 BP values than among patients with stage 1 or stage 2 BP values ($P = .004$, Cochran’s linear trend $P = .002$). No significant differences between the BP groups were noted in the prevalence of chest pain, dizziness, headache, focal neurological deficit, nosebleed, visual change, or headache complaints. No intergroup difference was noted in the prevalence of patients reporting at least one hypertension-associated symptom in their chief complaint.

In part 2, information regarding hypertension-associated symptoms was derived from patient interviews after prompting by research associates. Results are shown in Table 3. Sixty-eight percent of interviewed patients reported having one or more hypertension-associated symptoms at the time of arrival in the ED. No significant association between BP stage and the prevalence of any hypertension-associated symptom were noted. Further analyses revealed no significant intergroup differences in the prevalence of individual or aggregate hypertension-associated symptoms in the 24 h or 1 week before ED presentation.

4. Discussion

As many as 27% of adults in the United States are believed to have hypertension [11]. Reflecting the high prevalence of this condition, sustained BP elevation was noted in 29% of the adult ED patients in our study.
Although the majority of these patients have mildly elevated BP values, severely elevated BP is not uncommon in the ED. One large retrospective study in an urban ED documented stage 3 BP values in 2% of patients [12], whereas our prospective study found that 5% of adult ED patients met these criteria.

The assessment and management of patients with elevated BP have been extensively studied in primary-care environments. Evidence-based standards for classifying and treating hypertension have been established by the JNC [1]. In their sixth report (JNC-VI), the authors define hypertension staging criteria that are widely cited in the medical literature and commonly used in assessing ED patients. The most recent JNC report (JNC-7) no longer employs this staging system [13].

The JNC authors specify that their hypertension management guidelines are intended for primary-care clinicians in assessing and managing BP elevation in patients without acute illnesses [2]. Extrapolation of the JNC guidelines to the ED setting may not be appropriate. ED patients often have acute illnesses or injuries that may elevate or depress their BP. It is generally not practical or possible to measure the BP according to JNC’s rigorous standards, and therapeutic decisions are often made without the repeated BP assessments advised in the JNC guidelines. Despite widespread adoption of the JNC-VI’s BP staging and management recommendations by emergency medicine textbooks and other guidelines for ED care [3,4], these guidelines were neither derived nor validated for use in the ED setting [14].

The purpose of this study was to determine if symptoms putatively regarded as associated with hypertension are more prevalent among ED patients with greater degrees of BP elevation. Failure to detect a relationship between symptom prevalence and BP value would bring into question the validity of ascribing these symptoms to BP elevation. An unprompted complaint of dyspnea was, in fact, found to be more common among patients with greater degrees of BP elevation. However, no other complaints potentially suggestive of acute cardiovascular, cerebral, or visual dysfunction differed significantly between patients in any BP group.

Patient interviews revealed that symptoms often attributed to hypertension are highly prevalent among ED patients with elevated BP. When patients with elevated BP were prompted for symptom presence, more than two thirds reported having at least one symptom potentially attributable to hypertension. No single symptom was more frequent among patients with greater degrees of BP elevation, and the overall prevalence of any hypertension-related symptom did not differ between BP groups.

Hypertensive emergencies are defined as relatively rare situations requiring immediate reduction in BP to prevent or limit acute damage to the brain, heart, eyes, and kidneys [1-7,15]. Many authorities suggest that in the setting of significantly elevated BP, hypertension-associated symptoms should be presumed to reflect acute end-organ injury and that the presence of these symptoms distinguishes hypertensive emergency from a non-critical hypertensive urgency [1,6,7]. These criteria may be overly simplistic and would lead to the diagnosis of hypertensive emergency in the majority of ED patients with elevated BP in our study. Our findings are consistent with an Italian study noting hypertension-associated symptoms in 28% of ED patients with severe BP elevation [16]. The authors of that study concluded that hypertensive emergencies are common events in the ED population. An alternative explanation, however, is that hypertension-related symptoms are so nonspecific as to make their presence alone inadequate to diagnose hypertensive emergency.

4.1. Limitations

We studied only ED patients with elevated BP. Stronger conclusions regarding the association of hypertension-associated symptoms and BP magnitude require that symptom prevalence be determined in ED patients with normal BP values. It is possible that some study patients would be normotensive under other circumstances or that BP values would normalize if further measurements were taken in the ED [17].

As previously mentioned, the BP measurement standards used in the JNC reports were not intended for the ED and are impossible to strictly apply in this setting. We did not train the ED staff in the BP measurement techniques advised in the report. Despite the flaws inherent in applying the JNC BP staging criteria to ED patients, these criteria are widely used in this setting, frequently cited in the emergency medicine literature, and familiar to emergency physicians. We believe our BP measurement practices are typical of those in other EDs.

This study was conducted in a single, urban, academic ED, and the majority of our subjects were African American. Our findings may not be generalizable to other ED settings or other populations. Larger multicenter studies are needed to confirm these results.

Recall bias may play a significant role in our calculation of symptom prevalence. It is possible that prompting by the research associates in part 2 of the study may have caused patients to overstate the prevalence of hypertension-associated symptoms. This bias is unlikely to have affected the prevalence of hypertension-associated symptoms in part 1 which were derived from the medical record. Because we relied on patient-reported complaints rather than physician diagnoses, it is possible that some complaints, such as chest pain, were not cardiovascular in etiology and that some patients would not be classified as having symptomatic hypertension.

Our failure to detect significant intergroup differences in symptom prevalence may have been caused by type II error. Epistaxis was the chief complaint of only 0.3% of subjects, and retrospective power calculations show that more than 7000 subjects in each BP group would have been necessary
to detect a doubling in prevalence of this complaint. A recent prospective study has, in fact, demonstrated active epistaxis to be more common among patients with greater BP values [18]. For the more common symptoms noted in part I of our study, such as dyspnea, chest pain, and headache, our sample size was sufficient to detect an absolute intergroup difference in symptom prevalence of about 15% with a power of 80%. Other than the prevalence of dyspnea, we noted no trends toward intergroup differences in symptom prevalence.

4.2. Conclusions

We conclude that the symptoms commonly regarded as being associated with hypertension are highly prevalent among ED patients with elevated BP. The majority of patients with elevated BP values report having at least one such symptom, and there is no relationship between the prevalence of most symptoms and the patient’s BP stage. Physicians should exercise caution when attributing symptoms to BP elevation in this population.

Acknowledgment

The authors thank Jane Prosser, MD, for valuable assistance with data analysis and entry; to Kathleen Hatala, BSN, for assistance with data management; and to Christine Shields, RN, for supervising data acquisition.

References

A comparison of emergency department versus inpatient chest pain observation units

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Received 25 January 2004; accepted 5 March 2004

Abstract US hospitals use observation units (OUs) for safe and cost-effective management of low-risk to moderate-risk patients presenting to the ED with chest pain. This study retrospectively compared the utility of an ED observation unit (EDOU) with an inhospital observation unit (IHOU) for chest pain at the same institution. A 5-month period during which patients with chest pain were admitted to the EDOU was compared with a 5-month period during which patients with chest pain were admitted to the IHOU. During the 5-month EDOU period, 440 (36.9\%) of 1190 patients with chest pain presenting to the ED were admitted for observation. During the IHOU period, 973 (69.3\%) of 1404 patients with chest pain presenting to the ED were admitted for observation (\(P < .0001\)). Fewer patients with chest pain were converted to full inpatient admission from the EDOU, 35 (7.9\%) of 440, when compared with the IHOU, 187 (19.2\%) of 973 (\(P < .0001\)). Mean cost for each patient was US$889.87 (95\% CI 862.8-916.9) versus US$1039.70 (95\% CI 991.7-1087.7) for each IHOU patient. We conclude that the EDOUs are more cost-effective than IHOUs for management of low-risk to moderate-risk patients with chest pain.
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1. Introduction

Observation units (OUs) have become increasingly common in US hospitals during the past decade. OUs offer numerous advantages to patients, EDs, and hospitals including rapid diagnosis and treatment of low-risk but potentially life-threatening complaints, cost-effective care and evaluation, improved reimbursement for hospitals, and generation of revenues for EDs [1]. The organization and management of patients admitted for observation services are variable in the United States. The most common models are ED-based OUs (EDOU), dedicated inhospital OUs (IHOU), and admitting patients to “observation status” on regular hospital floors, without having a dedicated space for an OU. Numerous studies have evaluated the cost-effectiveness of OUs, improvements in clinical processes, impact on decisions to admit patients from the ED, effectiveness of various...
2. Methods

This study was a retrospective observational study of all ED patients admitted with chest pain to an EDOU and an IHOU at a university hospital with 75 000 ED visits annually. Data from two 5-month periods were collected from each OU with similar inclusion and exclusion criteria. Data were collected from the EDOU between November 1997 and March 1998 and from the IHOU between May 1998 and September 1998. During the EDOU period, the EDOU was the only OU available for eligible patients with chest pain, and during the IHOU period, the IHOU was the only OU available for eligible patients with chest pain. Both the EDOU and IHOU were open 24 hours a day, 7 days a week. For both OUs, the decision to admit an ED patient with chest pain to the OU was made by a board-certified emergency physician after ED evaluation. The group of emergency physicians did not change during the two 5-month periods.

Patients were eligible for admission to either OU if they were presented to the ED with chest pain, had a nondiagnostic electrocardiogram, and were determined by the ED physician to have low to moderate suspicion for acute coronary syndrome or myocardial infarction (MI). The ED physician made this determination of risk during the ED evaluation by assessing the likelihood of coronary artery disease as well as assigning a risk category for each patient, using the Canadian Cardiovascular Society classification system [7].

Patients were not eligible for admission to either OU if they were at high suspicion for MI or unstable angina, if they were having an MI, or if their initial set of cardiac enzymes was positive. Patients were also not eligible for OU admission if their chest pain was determined by the ED physician not to be of cardiac origin. Patient management in the OU included monitoring, serial cardiac enzymes, and evaluation by a cardiologist once an MI had been ruled out. Patients then usually underwent cardiac stress testing before discharge, a decision made by the cardiologist. Patients were admitted to the hospital (regular admission) during their OU evaluation if they developed electrocardiogram changes, had positive cardiac enzymes (ruled in for an MI), had a positive stress test, became unstable during their OU evaluation, or were felt by the cardiologist to be at high likelihood for MI or unstable angina.

During the first period, ED physicians managed the EDOU; in the second period, non-ED physicians managed the IHOU. For each OU, we analyzed and compared the total number of admissions, percentage of total ED visits for chest pain admitted to each, the rate of conversion to inpatient admission, age, sex, and costs using $\chi^2$ and 2-sample $t$ tests.

Hospital charge was used as a proxy to compare costs of caring for EDOU and IHOU patients. Information on ED and hospital charges was obtained and calculated using the hospital’s cost accounting system. The total charge was derived by identifying the resources used by each patient as represented on their final bill (including room charges for observation stay, tests, medications, etc) and multiplying each of these resources by a constant called the per-unit cost (the cost to the hospital to provide each resource). Per-unit costs include labor (staff), supplies, equipment depreciation, and an allocation of hospitalwide indirect costs.

This study was retrospective, and after review was considered exempt by the hospital’s institutional review board.

3. Results

During the 5-month EDOU period, 440 (36.9%) of 1190 patients with chest pain presenting to the ED were admitted for observation. During the IHOU period, 973 (69.3%) of 1404 patients with chest pain presenting to the ED were admitted for observation ($P < .0001$). The patient groups admitted to the EDOU and the IHOU were similar. There was no significant age or sex differences in admission to either the EDOU or the IHOU. Fewer patients with chest pain were converted to full inpatient admission from the EDOU, 35 (7.9%) of 440, when compared with the IHOU, 187 (19.2%) of 973 ($P < .0001$). Mean charge for each patient was US$889.87 (95% CI 862.8-916.9) versus US$1039.70 (95% CI 991.7-1087.7) for each IHOU patient.

4. Discussion

This study examined whether patients with signs and symptoms consistent with acute chest pain could be cared for in a more cost-effective way in an EDOU as compared with an IHOU. The inpatient cost of patients ultimately determined to have noncardiac chest pain is high [8]. The challenge for emergency physicians is to identify and risk-stratify low-risk patients with chest pain who require only a limited (and less costly) evaluation at a time when emergency physicians are seen as the leading source of malpractice suits for failure to diagnose and treat acute MI [9].

Chest pain is one of the most common complaints of patients presenting to an ED, comprising up to 5% of all visits, yet only 10% to 15% of those will have an acute MI [10]. Since 33% of patients with acute chest pain admitted to the hospital do not have cardiac disease, it is these...
groups that are ideal candidates for an EDOU [11]. Some estimates are that chest pain OUs save about US$2000 per patient or cost about 20% to 50% less than an inpatient admission [12]. Nationwide that would translate to 1 billion dollars saved if 10% (500,000) of ED patients with chest pain were admitted to an OU as opposed to a regular inpatient admission. Our data suggest that costs are even further reduced if patients with low-risk chest pain are admitted to an EDOU when compared with an IHOU. In addition, as a smaller percentage of EDOU patients were converted to full inpatient admissions, patient care was more efficient, and low-risk patients with chest pain occupied fewer inpatient beds.

The reasons for the differences identified in this study are not explained by the study results. However, one likely possibility is that during the EDOU phase, all patients and caregivers (ED physicians and nurses) were in the same geographic location and were focused on completing the patient workup and disposition in the observation period. During the IHOU phase, all patients and caregivers (non-ED physician and nurses) were not in the same geographic location, and completing the patient’s workup and disposition during the OU period may have been more difficult.

As a retrospective observational study, this study is subject to some limitations, including relying on proper documentation. During the EDOU phase of the study, ED physicians may have admitted a greater number of low-risk patients to the EDOU. During the IHOU phase, ED physicians may have admitted more moderate-risk or high-risk patients to the IHOU. As a result, it is possible that a misclassification bias was introduced during the two 5-month study periods, limiting the interpretation of study results.

In retrospect, this investigation could have been improved if the study period for both the EDOU and IHOU phases of this study had been longer, allowing more data collection; in addition, using a different mechanism to approximate true costs instead of using as a proxy for costs may yield more reliable data. Finally, prospective randomized trials with strict entry criteria comparing length of stay, costs, and outcomes for EDOU and IHOU patients presenting with chest pain of suspected cardiac origin would be useful to confirm our study findings.

In summary, mean cost for patients admitted to the EDOU for chest pain was significantly less than IHOU despite a higher mean EDOU patient age. Patients with chest pain during the IHOU period were more likely to be admitted both for observation as well as be converted to inpatient status. This study suggests that at least from a cost perspective, EDOUs are a cost-effective, medically sound alternative to stratifying patients presenting with low-risk to moderate-risk chest pain.

References
A randomized, double-blind study comparing morphine with fentanyl in prehospital analgesia

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Received 26 August 2003; accepted 5 March 2004

Abstract

Study Objective: The aim of this study was to compare, by a randomized double-blind method, morphine (M) and fentanyl (F) in a prehospital setting.

Methods: Consecutive patients with severe, acute pain defined as a visual analog scale score (VASS) of 60/100 or higher were included. The M group received an initial intravenous M injection of 0.1 mg/kg then of 3 mg every 5 minutes. The F group received an initial intravenous F injection of 1 l g/kg then of 30 l g every 5 minutes. The goal of analgesia was a VASS of 30/100 or lower. The end point was the VASS measured 30 minutes after initial administration (VASS[T30]).

Results: There were 26 patients included in the M group and 28 in the F group. Initial VASS(T0) and VASS(T30), mean (95% CI), were 83 (78-88) and 40 (28-52) in the M group and 77 (72-82) and 35 (27-43) in the F group (P = NS). Sixty-two percent of patients in the M group described analgesia as excellent or good vs 76% of those in the F group who did (P = NS). There were no differences in the incidence of side effects in the 2 groups.

Conclusion: This study demonstrates that M and F were comparable in treating severe, acute pain in a prehospital setting during the first 30 minutes in spontaneous breathing patients.

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1. Introduction

Recommended initial analgesia of patients with severe, acute pain, defined as a visual analog scale score (VASS) of 60/100 or higher, in a prehospital setting in France consists of the administration of opioids by the medical staff of mobile intensive care units. International guidelines generally recommend morphine (M) and fentanyl (F) as opioid medications [1-4]. The intravenous administration of M is usually considered as the gold standard for postoperative acute pain relief because of its rapid transport from the blood to target tissues after intravenous injection, its long-lasting analgesic effect without any plateau, and its well-known pharmacokinetics [2,3]. Nevertheless, the short-acting opioid F might be preferable to the traditional long-acting M for prehospital analgesia because of its even faster onset of action and shorter duration than M [3,5,6]. To date, there is no study, to our knowledge, comparing the clinical efficacy of F vs M in a prehospital setting.

The aim of this study was to determine whether F is more or less effective than M during the first 30 minutes of prehospital treatment of patients with severe, acute pain.

2. Materials and methods

We performed a prospective, multicenter, randomized, double-blind controlled study. The trial was coordinated by the University Hospital of Avicenne (Bobigny, France). Five prehospital emergency services employing mobile intensive care units and located in urban areas participated in this study. In France, management of out-of-hospital medical emergencies is the responsibility of the Service d’Aide Médicale Urgente. Mobile intensive care units are staffed by an attending emergency physician, a nurse anesthetist, and an EMT.

The human subjects committee of the Robert Ballanger Hospital (Aulnay, France) approved this study, and all patients provided written informed consent. Patients were recruited between January 1, 2001, and December 30, 2002.

A table of random numbers determined the randomization sequence, using a restricted randomization scheme to ensure roughly equal numbers in each group. Group assignments were sealed in opaque envelopes and opened sequentially by the investigators. Eligible patients were randomly allocated to receive either M (M group) or F.

![Fig. 1](image-url)  The study protocol involving administration of F vs M.
(F group) intravenously. Patients were asked to assess the intensity of their pain by identifying a VASS upon inclusion (VASS [T0]) and then every 5 minutes using a tool with a 100-mm ruler and a marker that patients move to the point indicating their intensity of pain. The VASS was presented as a horizontal line on which each patient’s pain intensity is represented by a point between the extremes of “no pain at all” and “worst pain imaginable.” Patients were eligible for inclusion if they presented a severe, acute pain defined by a VASS of 60/100 or higher and with age between 18 and 70 years. Exclusion criteria included the presence of chronic respiratory, renal, or hepatic insufficiency, known opioid allergies, treatment of chronic pain or treatment with opioids, incapacity to understand the VAS, acute hemodynamic, respiratory, or neurological compromise, pregnancy, or indication for local or regional analgesia. Patients who had already received an opioid analgesic (either by self-administration or by another physician in attendance) were also excluded. Intravenous analgesia was given and titrated to the pain score every 5 minutes. The drugs were used in equipotent analgesic doses administered from syringes of similar appearance prepared by a nurse anesthetist who was not otherwise involved in the study. Each syringe contained either 20 mL of 1 mg/mL M (M group) or 20 mL of 10 μg/mL F (F group).

An independent physician-observer blinded to the analgesic treatment groups did all assessments of patients. The first volume administered was 1 mL per 10 kg (ie, 0.1 mg/kg of M [M group] or 1 μg/kg of F [F group]) followed by additional volumes of 3 mL until pain relief was obtained, as defined by a VASS of 30/100 or lower. The doses were chosen on the basis of a pilot experiment showing that administration of lower doses of M (0.05 mg/kg for the initial bolus), as proposed by French guidelines, was insufficient [1]. The study protocol is represented in Fig. 1.

The end point of the study was the VASS at T30. Patients were asked to assess the intensity of their pain every 5 minutes until arrival at a hospital. All VASSs were recorded at T0, T10, T20, and T30. Thirty minutes (T30) after the first injection (T0), overall patients’ and investigators’ satisfaction with analgesia (pain relief classified as excellent, good, mild, or weak) was recorded. The safety evaluation included monitoring of blood pressure, heart rate, respiratory rate, and oxygen saturation by pulse oximetry and a sedation scale (0, patient awake; 1, patient with intermittent sleeping; 2, patient sleeping, awakened by verbal stimulation; 3, patient sleeping, awakened by tactile stimulation; 4, patient not aroused by stimulation). The presence of nausea, vomiting, dizziness, itching, drowsiness, and bradypnea was likewise recorded. These data were recorded at T0 and T30. The presence of side effects resulted in an interruption of opioid administration.

The Student t test was used for comparison of quantitative variables. The aim of postoperative or prehospital pain relief was a VASS of 30/100 or lower, and SD was estimated to be approximately 15/100 [7]. For reaching

### Table 1 Baseline characteristics of patients from groups M and F

<table>
<thead>
<tr>
<th></th>
<th>M group (n = 26)</th>
<th>F group (n = 28)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age [y (SD)]</td>
<td>40 (13)</td>
<td>45 (13)</td>
<td>NS</td>
</tr>
<tr>
<td>Sex ratio (F/M)</td>
<td>3:23</td>
<td>6:22</td>
<td>NS</td>
</tr>
<tr>
<td>Mean BMI [kg/m² (SD)]</td>
<td>24 (3)</td>
<td>24 (4)</td>
<td>NS</td>
</tr>
<tr>
<td>Cumulative volume administered at</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T0 [mL (SD)]</td>
<td>7 (1)</td>
<td>7 (2)</td>
<td>NS</td>
</tr>
<tr>
<td>T30 [mL (SD)]</td>
<td>16 (5)</td>
<td>15 (6)</td>
<td>NS</td>
</tr>
<tr>
<td>Etiology of pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trauma [n (%)]</td>
<td>19 (73)</td>
<td>14 (50)</td>
<td>NS</td>
</tr>
<tr>
<td>Nontrauma [n (%)]</td>
<td>7 (27)</td>
<td>14 (50)</td>
<td></td>
</tr>
<tr>
<td>Comorbidity [n (%)]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>1 (4)</td>
<td>1 (4)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1 (4)</td>
<td>3 (11)</td>
<td></td>
</tr>
<tr>
<td>Diabetes/dyslipidemia</td>
<td>2 (8)</td>
<td>3 (11)</td>
<td></td>
</tr>
</tbody>
</table>

BMI indicates body mass index.

### Table 2 Comparison of different analgesia parameters between the M group and F group

<table>
<thead>
<tr>
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<th>M group (n = 26)</th>
<th>F group (n = 28)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side effects [n (%)]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>10 (38)</td>
<td>10 (36)</td>
<td>NS</td>
</tr>
<tr>
<td>Emesis</td>
<td>3</td>
<td>6</td>
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</tr>
<tr>
<td>Dysphoria</td>
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<td>1</td>
<td></td>
</tr>
<tr>
<td>Pruritus</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Sedation score = 2 [n (%)]</td>
<td>3 (11)</td>
<td>0 (0)</td>
<td>NS</td>
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<tr>
<td>Patient satisfaction:</td>
<td></td>
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<tr>
<td>excellent/good (%)</td>
<td>62</td>
<td>76</td>
<td>NS</td>
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<td>Duration from T0 to hospital [min (mean SD)] at</td>
<td></td>
<td></td>
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<tr>
<td>T0</td>
<td>39 (15)</td>
<td>34 (10)</td>
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<tr>
<td>T30</td>
<td></td>
<td></td>
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<tr>
<td>Heart rate [beats/min (mean SD)] at</td>
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<tr>
<td>T0</td>
<td>81 (16)</td>
<td>84 (20)</td>
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<td>T30</td>
<td>82 (16)</td>
<td>81 (17)</td>
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<td>Respiratory rate [beats/min (mean SD)] at</td>
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<tr>
<td>T0</td>
<td>19 (4)</td>
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</tr>
<tr>
<td>T30</td>
<td>16 (3)</td>
<td>17 (5)</td>
<td></td>
</tr>
<tr>
<td>SpO₂ [% (mean SD)]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T0</td>
<td>98 (2)</td>
<td>98 (2)</td>
<td>NS</td>
</tr>
<tr>
<td>T30</td>
<td>98 (3)</td>
<td>98 (3)</td>
<td></td>
</tr>
<tr>
<td>Other treatments</td>
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<td></td>
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</tr>
<tr>
<td>administered (n)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>4</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>4</td>
<td>5</td>
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</table>
a VASS difference higher than 14/100 in favor of F, the appropriate sample size using an α error of .05 and a β error of .10 was calculated with the Casagrande et al formula [8]. A minimum of 26 patients for each group should be included to see a difference of 14 mm between groups. We chose to include 30 patients in each group to increase the power of this study. All statistics were performed using Statview Software (StatView version 5, Abacus Concepts, SAS Institute, Berkeley, Calif). A P value of <.05 was considered statistically significant.

3. Results

Sixty patients were included between January 1, 2001, and December 31, 2002. Four patients in the M group were withdrawn from the analysis because of the use of a hypnotic (propofol; n = 1), thrombolysis for acute myocardial infarction (AMI; n = 1), and cases in which the protocol design was not respected (n = 2). In the F group, 2 patients were withdrawn because of the use of a hypnotic (propofol) of 1 patient and because the protocol design was not respected by the other. Thus, data from 54 patients were analyzed, 26 in the M group and 28 in the F group. The 2 groups were similar with respect to demographics, duration of intervention, and quantities of F and M administered (Table 1). Eight patients (15%) were older than 60 years, without a significant difference between groups. Initial mean (95% CI) VASS(T0) and VASS(T30) were 83 (78-88) and 40 (28-52) in the M group and 77 (72-82) and 35 (27-43) in the F group ($P =\ NS$) (Fig. 2). There were no differences between groups with regard to blood pressure, heart rate, respiratory rate, or oxygen saturation at T0 and T30 (Table 2). Evolution of mean (95% CI) VASS variation ($\Delta$VASS[Tx], defined as VASS[T0] – VASS[Tx]) is shown in Fig. 3. At T30, mean (95% CI) change in VASS was 45 (34-56) in the M group and 42 (32-52) in the F group ($P =\ NS$). Sixty-two percent of patients in the M group described analgesia as excellent or good vs 76% of those in the F group who did ($P =\ NS$). The incidence of side effects was comparable in both groups (Table 2). The number of patients with a VASS of 30 or lower is shown in Fig. 4. At T30, 65% of the patients in the M group had a VASS of 30 or lower vs 57% of those in the F group ($P =\ NS$).

4. Discussion

In this study, M and F appeared to be clinically comparable for treatment of severe, acute pain in a prehospital setting with no significant difference in pain intensity between groups 30 minutes after the beginning of administration. In addition, we found no difference in the incidence of side effects. The efficacies of the 2 molecules are comparable because we found that 65% of patients from the M group and 57% from the F group reached a VASS(T30) of 30/100 or lower. A few studies have shown intravenous nitroglycerin (NTG) to be modestly effective in relieving chest pain in AMI or acute coronary syndrome [9,10]. In our study, only 7 patients (3 in the M group and 4 in the F group) received intravenous NTG during AMI or acute coronary syndrome. Even if NTG administration interfered with the analgesia procedure, the comparison between the 2 randomized groups was not altered. Two patients received only sublingual spray NTG, which was not effective.

Obtaining high-quality analgesia in prehospital patients with severe pain is an important treatment objective not only for psychological but also for physiological reasons. It is generally agreed that acute pain results in the activation of
the sympathetic nervous system, which may adversely affect cardiac and respiratory functions [11,12]. Nonetheless, most studies have shown that acute pain treatment in an emergency setting is insufficient [7,13,14]. We hypothesized that this might be explained by the nature of the opioid used. One study compared M and alfentanil in a prehospital setting in patients with acute ischemic-type chest pain [15]. In the alfentanil group, onset of pain relief was faster and analgesia was more effective than in the M group throughout the first 15 minutes. The follow-up period was limited to 15 minutes. In this study, initial pain was severe or mild and there was no opioid titration [15]. Because alfentanil’s duration of action is very short, it is not considered to be ideal for prehospital acute pain [1,6].

The ideal prehospital opioid should have a rapid onset of action, providing rapid control of pain with a duration of action long enough to avoid repeated injections. Fentanyl, a very lipid soluble with a faster onset of action (3 minutes) than M (whose onset of action begins 10 or 15 minutes after injection) should permit faster pain relief [5,6,16]. The onset time of analgesia was significantly faster for 10 mg M compared with 5 mg F in a postoperative setting [17]. Nevertheless, in clinical practice, M administration is rapidly efficacious with an onset of action allowing rapid pain relief [18,19]. In our 30-minute study, no significant differences were detected between the opioids at any time. Another potential advantage of M is its long duration of action in comparison with other opioids. Its duration is 3 to 4 hours vs 30 minutes to 2 hours for F although the elimination half-lives are 114 minutes and 185 to 220 minutes, respectively [4]. Morphine and F were compared during postanesthesia recovery [20]. This study showed M and F to be comparable in treating the first 40 minutes of postoperative pain after ambulatory procedures. Morphine produced sustained analgesia, whereas patients receiving F required additional oral analgesia after the first 40 minutes. However, there were more side effects with M [20]. The results of this study are not easily extrapolated to the situation of emergency patients because postoperative patients have already received sedation and analgesia during surgery.

On the other hand, a few studies have shown that F and M, in clinical practice, are safe in emergency department use or in a prehospital setting when they are each used alone [21,22]. But one study showed that F (administered at 2 µg/kg) alone produced significant hypoxemia in half of the subjects and deep depression of ventilatory response to CO2 [23]. In the same study, midazolam (0.05 mg/kg) and F in combination significantly increased the incidence of hypoxemia (11 of 12 subjects) and apnea (6 of 12 subjects) [23]. Wright et al [24] showed respiratory depression in 0.5% emergency department patients who received midazolam in combination with F.

Our results suggest that administration of opioids (M or F) is safe because there were no major side effects (respiratory depression).

This study demonstrates that F is not more efficacious than M in treating severe, acute pain in a prehospital setting. No difference in the onset of analgesia was established in our observations. Both opioids are safe during the first 30 minutes that the doses are administered. The incidence of side effects was not different between the 2 groups and no severe complications appeared although relatively high doses were used.

References


Unreliability of reported tetanus vaccination histories

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Received 5 March 2004; accepted 6 March 2004


Abstract Our study questioned the reliability of patients’ reported tetanus immunization histories. One thousand patients at an urban teaching hospital were queried regarding their tetanus immunization status. Of the 377 patients who initially asserted having had a tetanus vaccine in the last 5 years, 98 (26%) were confirmed, either by further history taking or chart review, not to have received tetanus immunization. It appears that there is a sizeable percentage of patients who falsely report their tetanus immunization status.

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1. Introduction

Surveys of emergency department patients suggest that 1% to 6% of all persons in the United States who receive medical care for injuries that can lead to tetanus do not receive prophylaxis as recommended by the Advisory Committee on Immunization Practices [1]. However, it has been shown that, in a primary care setting, mothers do not reliably recall the immunization statuses of their children and tend to falsely estimate the number of vaccines their children had received [2,3]. No study to date has addressed the reliability of reported tetanus histories in an adult acute care setting.

The initial history obtained from patients concerning their last tetanus vaccination is most often taken at face value. Further elaboration frequently reveals that what patients had initially thought was a tetanus vaccine was, in fact, some other needle injection (purified protein derivative [PPD], hepatitis booster, influenza vaccine, etc). Overconfidence in an unreliable history may lead to nonimmunization of patients at risk for tetanus. We sought to objectively evaluate the reliability of reported tetanus immunization histories.

2. Materials and methods

We recently had our residents at an urban teaching hospital with an ED census of more than 95 000 administer a survey to 1000 patients regardless of their presenting complaints. After obtaining informed consent, patients were asked, “When was your last tetanus vaccine?” Patients were classified into 3 categories based on their response. First were those patients who had no recollection as to when their last vaccination was. Second were those who recalled that their last vaccine was administered more than 5 years ago. Third were those who asserted that their last vaccine was administered within the last 5 years. The first 2 groups were excluded from further questioning because, in an acute
care setting, the patients in these groups would be vaccinated if clinically indicated.

Those patients who asserted that their vaccines were taken within the last 5 years were next asked, “Why did you receive the vaccine?” Their response was considered accurate (1) if they gave a history of vaccine administration in response to a break in the skin or (2) if the history was confirmed upon further review of their medical records. Conversely, their response was considered inaccurate if further history or review of their medical records indicated that the vaccination they received was not for tetanus immunization. The response was considered indeterminate if further history and review of their medical records were equivocal.

The internal review board of our hospital approved all aspects of this study.

3. Results

Of the 1000 patients queried, 623 patients had no recollection of when they had their last tetanus vaccine or recalled that they received it more than 5 years ago. Of the 377 who recalled that their vaccination was administered within the last 5 years, 143 patients’ vaccinations were verified to be for tetanus immunization by a history of a skin break. Of the 377, 52 were determined by further history as having received an injection other than tetanus (see Table 1). The remaining 182 were indeterminate by initial history and further investigation into their medical records was therefore required. Of these 182, 22 were confirmed to have been administered a tetanus vaccine according to their medical records. Of 182, 46 were determined to have been administered injections other than tetanus or were given no vaccine at all (see Table 2). The remaining 114 were indeterminate because of inability to access their medical records. Of the 377 who initially asserted having had a tetanus vaccine in the last 5 years, 165 (43.8%) were confirmed to have actually received a tetanus vaccine, 98 (26.0%) were confirmed not to have received a tetanus vaccine, and 114 (30.2%) were indeterminate.

4. Discussion

Our data support that histories obtained from patients concerning their tetanus statuses are unreliable. Twenty-six percent of patients who initially stated that they have received a tetanus vaccination within the last 5 years were confirmed to have never received prophylaxis. Further clarification of those histories may be helpful in ascertaining patients’ true tetanus statuses. Taking a few moments to gain more accurate information would facilitate appropriate vaccination in those patients who may otherwise miss the opportunity to be properly vaccinated. However, there still exists a substantial subset of patients in whom additional history taking will not be able to delineate their true tetanus statuses.

A major limitation to our study may be external validity. In other settings outside an urban teaching hospital, patients may be more informed about their vaccination history, possibly because they have received more consistent medical care by a primary physician. We have seen that most patients who falsely reported their tetanus status received the injection in confusion with PPD administration. Our hospital’s population may receive a larger amount of PPDs than in other settings because of requirements set forth by entities such as drug detoxification programs and the United States penal system, thus allowing for more confusion between the 2 injections in our population. Further studies in other clinical settings need to be undertaken to see if results similar to ours would be found.

Another limitation is the large number of indeterminate histories. However, even if most of these indeterminate histories were subsequently found to ascertain tetanus vaccination, it still does not alter the fact that a significant number of patients (26%) did in fact convey false information in reporting their tetanus status. Finally, there still exists the possibility that vaccines were administered but not documented in the patients’ medical records.

5. Conclusions

It appears that there is a good percentage of patients who falsely report their tetanus immunization status. Much in the same manner that it has become commonplace to ask the details and nature of a patient’s stated drug “allergy” to determine whether it is true or not, we feel that the emergency physician should ascertain through further questioning the true nature of a patient’s tetanus status and not base important health decisions upon inaccurate data.

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<thead>
<tr>
<th>Table 1</th>
<th>Injections mistaken for tetanus after further history</th>
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<td>PPD</td>
<td>29</td>
</tr>
<tr>
<td>Analgesics</td>
<td>8</td>
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<tr>
<td>Flu vaccine</td>
<td>6</td>
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<tr>
<td>Hepatitis vaccine</td>
<td>3</td>
</tr>
<tr>
<td>Asthma treatment</td>
<td>3</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>2</td>
</tr>
<tr>
<td>Rabies vaccine</td>
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<tr>
<td>Total</td>
<td>52</td>
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<table>
<thead>
<tr>
<th>Table 2</th>
<th>Injections mistaken for tetanus after records reviewed</th>
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<td>PPD</td>
<td>12</td>
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<tr>
<td>Hepatitis vaccine</td>
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</tr>
<tr>
<td>Flu vaccine</td>
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</tr>
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<td>Analgesics</td>
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<td>Pneumovax</td>
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<td>No record of tetanus found in chart</td>
<td>28</td>
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<td>46</td>
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</tbody>
</table>
References

Retrograde endotracheal intubation: an investigation of indications, complications, and patient outcomes

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Received 5 February 2004; revised 29 March 2004; accepted 29 March 2004

Abstract The objective of this study was to review indications for retrograde endotracheal intubation (REI) and to examine outcomes and complications associated with this technique. We reviewed all intubations of adult emergency department patients over an 8-year period. A total of 1681 charts were reviewed with 313 excluded because of inadequate documentation of intubation. Of the 1368 remaining charts, we found that REI was attempted in 8 cases. Indications for REI included trauma (n = 4) and non-trauma-related respiratory distress (n = 4). Complications included inability to locate the cricothyroid membrane (n = 2), inability to pass the endotracheal tube through the vocal cords (n = 1), and procedure time of more than 3 minutes (n = 4). Retrograde endotracheal intubation was successful in securing the airway in 4 cases and unsuccessful in 4 cases. We found that although REI was attempted for trauma and non-trauma-related respiratory compromise, it was associated with multiple complications, and successful in only 4 of 8 cases.

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1. Introduction

Retrograde endotracheal intubation (REI) was first described by Butler and Cirillo in 1960 as an alternative to conducting unplanned preoperative tracheostomies [1] and has since been recommended for use as an alternative rescue airway procedure. Retrograde intubation kits are available in many North American academic emergency departments (EDs) (45% of 95 EDs responding to a survey) [2] and were found to be available in 8% of EDs (197 respondents) in a British survey [3].

Although REI continues to be cited by the American Society of Anesthesiology as an alternative strategy for dealing with the difficult airway [4,5], and REI equipment is frequently available to emergency physicians [2,3], there are no data available in the literature regarding indications for REI in the ED, or complications and outcomes of its use in the ED setting. We reviewed our experience with REI attempts in adult ED patients over a period of 8 years to identify the indications for the procedure, to examine complications of REI, and to report outcomes of REI attempts conducted by emergency physicians.

2. Methods

Our hospital is a level I trauma center which serves a region of approximately 3 million people and has an annual...
ED census of 53,000 patients. All adult patients intubated in the ED between January 1996 and August 2003 were included in the study. We chose these dates based on the ready availability of archived data from these dates at our hospital.

Patient charts were obtained by requesting from the Coding and Research Section of our hospital’s Health Information Management Department that all patients aged 18 or older who received ICD-9 codes for intubation (96.03, 96.04, and 96.05) in the ED from Jan 1996 to August 2003 be pulled for review. The charts then required manual searching for the specific cases in which retrograde intubation had been attempted. It could not be determined from the chart review whether the individual physicians attempting REI had received training for REI in a laboratory or if they had prior experience with REI.

An REI attempt was defined as puncture of the cricothyroid membrane with a needle in the anticipation of passing a guidewire through the needle. If investigators were not able to discern whether or not REI was attempted when reviewing a chart, the case was excluded from the study.

When details of the procedure were unclear, we spoke directly with the treating physician to see if they had reliable recall of the event. If details were not recorded or recalled, they were listed as “missing” on the data form.

We selected a priori 3 specific items of interest, which allowed us to achieve a focused retrieval of information from the patient’s medical records. First, we chose to study the indication for attempting to use retrograde endotracheal intubation as described by the treating physician in their report. We instructed chart abstractors to record the treating physician’s quoted reason for REI attempt.

The second item of interest was the complications encountered during REI attempts as described in the physician’s dictated report and in the patient’s hospitalization records. Potential complications that we listed on the data form were extracted from those found through a broad search of the literature and included hypoxemia [4], sore throat [4], cough [4], laryngospasm/bronchospasm [4], development of hematoma [6], incorrect site of tube placement [7], procedure time greater than or equal to 3 minutes [7], unsuccessful tube placement [7, 8], laryngeal fracture with permanent dystonia [7], pneumomediastinum [9], subcutaneous emphysema [9], infection [10], bleeding [5, 6, 11, 12], retained wire [13], nasal mucosal injuries as signified by the presence of epistaxis [12, 14], and “other.” If these complications were not documented in the medical record, they were assumed to be absent.

Finally, outcomes from REI attempts were examined in accordance with those recently listed by the American Society for Anesthesiology Task Force on Difficult Airway Management for other alternative airway approaches and included REI attempt time (if documented); whether REI was completed on first attempt (yes/no/not documented); and whether REI resulted in successful intubation (yes/no) [4].

### Table 1 Description of patients and outcome measures

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>Presenting problem</th>
<th>Indication for REI (direct quotation from records)</th>
<th>Complications of REI attempt</th>
<th>REI attempt time</th>
<th>REI successful on first attempt?</th>
<th>Outcome of REI attempt*</th>
</tr>
</thead>
<tbody>
<tr>
<td>18/M</td>
<td>Head and facial trauma</td>
<td>“vocal cords were obscured by blood”</td>
<td>None</td>
<td>2 min</td>
<td>Yes</td>
<td>Successful</td>
</tr>
<tr>
<td>59/F</td>
<td>Exacerbation of chronic obstructive pulmonary disease</td>
<td>“I couldn’t pass the ET tube through the cords”</td>
<td>None</td>
<td>3 min</td>
<td>Yes</td>
<td>Successful</td>
</tr>
<tr>
<td>57/M</td>
<td>Facial trauma</td>
<td>No direct quote</td>
<td>None</td>
<td>1 min</td>
<td>No</td>
<td>Not successful (orotracheal intubation)</td>
</tr>
<tr>
<td>55/M</td>
<td>Generalized trauma</td>
<td>“I was unable to intubate the patient”</td>
<td>Unable to locate cricothyroid membrane; hypoxia; prolonged REI attempt time; subcutaneous emphysema</td>
<td>5 min</td>
<td>No</td>
<td>Not successful (cricothyrotomy)</td>
</tr>
<tr>
<td>20/F</td>
<td>Cervical spine trauma</td>
<td>No direct quote “I couldn’t see vocal cords”</td>
<td>None</td>
<td>8 min</td>
<td>Yes</td>
<td>Successful</td>
</tr>
<tr>
<td>48/M</td>
<td>Pneumonia</td>
<td></td>
<td>Unable to locate cricothyroid via needle puncture on 3 attempts; hypoxia</td>
<td>Not recorded</td>
<td>No</td>
<td>Not successful (orotracheal intubation)</td>
</tr>
<tr>
<td>45/F</td>
<td>Exacerbation of chronic obstructive pulmonary disease</td>
<td>“There was a mass in the airway and I couldn’t pass the ET tube”</td>
<td>Unable to pass endotracheal tube through vocal cords</td>
<td>Not recorded</td>
<td>No</td>
<td>Not successful (orotracheal intubation)</td>
</tr>
<tr>
<td>57/M</td>
<td>Pulmonary edema</td>
<td>“I was unable to visualize vocal cords”</td>
<td>None</td>
<td>3 min</td>
<td>Yes</td>
<td>Successful</td>
</tr>
</tbody>
</table>

* Eventual successful technique listed in parentheses.
Manual data extraction from original medical records was required to identify cases in which REI had been attempted and to complete standardized data abstraction forms. The investigators trained abstracters in the completion of the standardized abstraction forms, and the principal investigator performed a blinded review of a random sample of 30% of all charts to determine reliability.

Two investigators independently completed standardized abstraction forms on each occurrence of REI that was identified, and percent agreement for study variables was determined.

Our hospital’s institutional review board approved this study through exemption from informed consent due to the use of archived data.

3. Results

The review of intubation ICD-9 codes identified 1681 patients. Upon investigator review, we then excluded 313 because there were not clear statements in the records confirming the method of intubation. Of the remaining 1368 records, we identified 8 in which REI was attempted (1 in every 170 intubations). The audit by the principal investigator of 410 (30%) of these records did not identify any discrepancies in the identification of REI (100% agreement).

The median age of the patient in whom REI was attempted was 48 years (range, 18-59), including 5 males and 3 females. The median time to intubation with REI was 3 minutes (not recorded in 2 cases). Patient descriptions, indications for REI, complications encountered during REI attempts, and outcomes (including times for REI attempts and whether REI was successful on first attempt) from REI attempts are seen in Table 1.

The 2 investigators concurred on classification of indications, complications, and outcomes for REI in all 8 cases (100% agreement).

4. Discussion

Since REI was first described 4 decades ago, many other alternative airway options have become available. Laryngeal mask airway, fiberoptic, and bougie-guided intubations are just a few of the techniques that emergency physicians currently use to rescue the difficult airway. Regardless of the availability of these more sophisticated tools, we are still occasionally faced with the patient whom we are unable to ventilate or intubate. As successful REI would potentially be less invasive than cricothyotomy or tracheostomy in these patients, we wanted to find out whether or not our experience with REI would support it being a reasonable intervention before cricothyotomy or tracheostomy.

There has only been 1 prospective study in which REI was conducted in patients, and, interestingly, this prehospital trial allowed for REI to be chosen as the initial airway option in selected situations [15]. Barriot and Riou reported that physicians of undisclosed REI training intubated 19 patients in less than 5 minutes and on first REI attempt in this study. The authors reported no complications associated with REI and concluded that REI was “easy to learn” and that “it should be developed for prehospital care of trauma patients [15].”

In our experience, the first REI attempt led to successful intubation in only 4 of 8 cases. Our procedure times were similar to Barriot and Riou’s, yet we experienced much less overall success with REI (50% compared with 100% in Barriot and Riou’s study). We experienced a variety of complications, including the inability to locate the cricothyroid membrane in 2 patients. One reason for the discrepancies between our findings and those of Barriot and Riou’s study could be that our 2 patient populations were very different. One third of the patients in Barriot and Riou’s study did not even receive initial conventional intubation attempts and may have been uncomplicated airways and easy intubations. The other two thirds of Barriot and Riou’s cases only received a single 1-minute conventional intubation attempt, whereas in all of our cases the patients failed conventional attempts, and the REI was being used as a last resort. Our cases, therefore, may be more representative of the difficult airway than Barriot and Riou’s. Another possibility is that physicians in Barriot and Riou’s study may have received specialized REI training just before the initiation of the study, whereas in our study, any REI training was likely to be much more remote to the actual REI attempts.

Other simulated REI studies have been conducted, including that of Van Stralen et al which reported that REI on a mannequin required a mean time of 70 seconds [16]. Stern and Spitzer found that inexperienced resident physicians’ mean time to complete REI on a cadaver was 56 ± 6 seconds [17]. Although simulated patient models are useful for teaching, they do not incorporate the dynamics of the stressful circumstances surrounding difficult airway management. Models are also problematic in that they do not reflect the anatomical airway nuances and bleeding complications that lead to difficult airway circumstances in real human patients.

Retrograde intubation has previously been evaluated for complications in one other case series [12]. Akinyemi described 12 cases using Water’s modified REI technique [18], which requires the use of Tuohy needle, epidural catheter tubing, and blunt hooks to fish the tubing out of the nose or oropharynx. As this technique has largely been replaced by standard REI kits since Akinyemi’s report, it is not surprising that the nature of the complications in this series differs from ours. It is interesting to note that 3 patients bled from the cricothyrotomy puncture site, and 2 from the nostrils in Akinyemi’s study, whereas in our study bleeding was not listed or recalled by physicians as a complication in any of the patients.

As there are no consensus guidelines for reporting ED airway management, it is difficult to compare REI directly
with other alternative airway strategies [19,20]. Additionally, federal mandates regarding emergency research will probably prevent a prospective comparison study of alternative airways from ever occurring [21,22]. Interestingly, when comparing our study to Bair’s retrospective study on cricothyrotomy, we found that it took more than 2 minutes to establish airway control with REI in 5 of 6 (83%) of those with time recorded) of our patients, whereas Bair et al found that cricothyrotomies took more than 2 minutes to perform in 17 of 50 patients (38%) [23]. McGill et al found that cricothyrotomy in the ED took longer than 3 minutes in only 4 of 38 patients (10%) [7]. When considered in this context, perceiving REI to be a better cosmetic alternative to cricothyrotomy probably does not justify its use.

Our study was limited by several important factors, namely, related to those typical of archival studies in general. We were forced to exclude 18% of the charts from the final analysis because the exact method of intubation was not clearly documented. It is possible that we may have missed REI occurrences because of nondocumentation; however, we believe that REI is unusual enough that clinicians would almost certainly mention it in their reports.

Complications may not have been fully recognized, detailed, and documented by treating physicians. This can occur for a multitude of reasons, including being unaware of the fact that complications were a result of REI attempts.

Timing of intubations is not always routinely documented at our facility, and it is possible that the times listed in Table 1 were inaccurate if nursing staff recorded the times after patients had been stabilized. This is often a necessary nursing routine in the face of prioritization regarding patient care activities. The REI attempt times which we recorded in Table 1 were therefore gleaned from the best available evidence in the chart. We felt that intubation attempt times were appropriate to mention despite potential documentation problems, as this outcome is of interest to clinicians who must decide which alternative airway techniques to learn, teach, and attempt [4,7].

Finally, because REI is not commonly used as a rescue airway method, we have a small sample size from which to draw our conclusions. Based on this limitation, we can only describe our experience and compare it to that of other authors.

5. Conclusion

Our 8-year experience found that REI was attempted in patients being intubated for trauma and non–trauma-related diseases. Retrograde intubation was attempted approximately once in every 170 intubations, was successful in half of the attempts, took a median of 3 minutes to complete (range, 1-8 minutes), and was associated with complications.

Acknowledgment

The authors would like to thank Renee Schroetlin, MD, for her assistance with this project.

References

Is the presence of *Trichomonas vaginalis* a reliable predictor of coinfection with *Chlamydia trachomatis* and/or *Neisseria gonorrhoeae* in female ED patients?

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Received 7 March 2004; accepted 30 April 2004

Abstract

**Objective:** The aim of the study was to determine if the presence of *Trichomonas vaginalis* (TV) is associated with an increased risk of coinfection with *Chlamydia trachomatis* (CT) and/or *Neisseria gonorrhoeae* (NG) in female patients presenting to the ED with symptoms consistent with a sexually transmitted disease (STD).

**Methods:** This was a retrospective observational study of female patients tested for the presence of TV by wet mount preparation in the ED from January 1998 through January 2001. Only patients that had the complete results of the wet preparation for TV, DNA probe for CT, and culture for NG were included.

**Results:** A total of 690 patients were enrolled in the study. Four hundred twenty (61%) patients tested positive for TV by wet mount preparation and 270 (39%) tested negative. Of the 420 TV-positive patients, 41 (9.8%) tested positive for NG, 35 (8.3%) tested positive for CT, and 16 (3.8%) for both. For the 270 TV-negative patients, 16 (5.9%) tested positive for NG, 91 (33.7%) tested positive for CT, and 16 (5.9%) for both. By \(\chi^2\) testing, there was a strong negative association between the presence of TV and coinfection with CT and/or NG. The presence of TV made it 2.9 times less likely to have coinfection with NG and/or CT.

**Conclusion:** The presence of TV in female patients presenting to the ED with symptoms consistent with an STD is not associated with an increased risk of coinfection with NG and/or CT.

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1. Introduction

Patients commonly present to the ED for symptoms related to a sexually transmitted disease (STD) [1,2]. Symptoms of STDs include vaginal discharge and irritation, dysuria, lower abdominal pain, and pelvic pain. The 2 most common bacterial STDs seen by emergency physicians...
(EPs) are caused by Chlamydia trachomatis (CT) and Neisseria gonorrhoeae (NG) [3]. Unfortunately, laboratory techniques for confirmation of the presence of these 2 organisms is normally by culture or DNA probe, resulting in a significant time delay for results. For this reason, most EPs are required to make the diagnosis of CT and/or NG infection based on history and physical examination alone, that is, empiric treatment based on clinical suspicion. Numerous studies have shown that only 25% to 50% of patients with proven NG or CT infection are effectively treated during their initial ED visit [2,4-7]. For those patients for which there is a low clinical suspicion, and subsequent positive test results, these patients are required to be called and informed to return to the ED for treatment or follow-up. Follow-up on these patients has been shown to be very problematic [4,5].

Another common STD, caused by Trichomonas vaginalis (TV), has been considered by some to be a useful marker for the presence of other STDs, like CT and NG [8]. Trichomonas infection is more commonly associated with symptoms such as vaginal discharge or vulvovaginal irritation than the more frequent, asymptomatic CT and NG infections [9,10]. If the presence of TV was found to be a reliable marker of concomitant infection with CT and/or NG, EPs might be more confident in empirically treating for these organisms. Similarly, the presence of TV might result in treatment of CT and/or NG in patients that otherwise would not have received such treatment on their initial ED visit.

The goal of our study was to determine if the presence of TV was associated with an increased risk of coinfection with CT and/or NG in female patients presenting to the ED with symptoms consistent with an STD.

2. Methods

This was a retrospective observational study of female patients tested for the presence of TV by wet mount preparation for the period January 1998 through January 2001 in the ED. Only patients that had the complete results of the wet mount preparation and CT and NG testing were included for review. This study was granted exemption from review by our school’s institutional review board.

All patients were seen in an urban hospital ED with an annual patient volume of 48,000. This ED serves as the primary training site for our fully approved PYG-I through PGY-III Emergency Medicine (EM) residency program. All patients were evaluated and managed by EM faculty or EM residents with EM faculty supervision.

For the period under study, it was routine to obtain endocervical/vaginal specimens for detection of TV, bacterial vaginosis, CT, and NG in women presenting to the ED with symptoms consistent with an STD.

The presence of Trichomonas was tested by wet mount preparation; infection was considered present if motile trichomonads were seen on microscopy. Analysis of the wet mount preparation was by trained technicians in our ED stat laboratory.

For detection of NG, culture of a specimen from the endocervical canal was taken by the physician and applied to a Thayer-Martin medium with carbon dioxide tablet. Cultures were performed by the Norfolk Public Health Department. The cultures are read at 36 hours and the results sent to the ED.

Testing for CT included obtaining a separate swab of the endocervical canal using a Gen-Probe PACE 2 (Gen-Probe, Inc., San Diego, Calif.) DNA probe and run in the main hospital laboratory. At our institution, the tests are processed once each day, and depending on the volume, each probe takes 4 to 8 hours to run.

For all patients meeting the inclusion criteria, the ED charts were reviewed for the results of the wet mount preparation, CT and NG tests.

Logistic regression analysis and \( \chi^2 \) testing were performed. In addition, odds ratios were determined. Statistical significance was said to be present at \( P < .05 \).

3. Results

A total of 690 patients were included in the study. Four hundred twenty (61%) patients tested positive on wet mount preparation for the presence of TV; 270 (39%) tested negative.

Of the 420 TV-positive patients, 41 (9.8%) tested positive for NG, 35 (8.3%) tested positive for CT, and 16 (3.8%) for both organisms. Three hundred twenty-eight (78%) patients tested negative for both CT and NG.

For the 270 TV-negative patients, 16 (5.9%) tested positive for NG, 91 (33.7%) tested positive for CT, and 16 (5.9%) for both. One hundred forty-seven (54.4%) patients tested negative for both CT and NG.

Analysis of the data found a strong negative association between the presence of TV and coinfection with CT and/or NG. Using odd ratios, we found that the presence of TV made it 2.9 times less likely to have a concomitant Chlamydia and/or Neisseria infection (\( P < .001 \)).

4. Discussion

Trichomonas infection (trichomoniasis) is considered an STD and is caused by the protozoan TV. It is very common among sexually active women, causing about 5 million new cases per year [11]. It is recovered from 66% to 100% of female partners of infected men and seen in 22% to 80% of male sexual partners of infected women [9]. It is normally sexually transmitted, but can be acquired nonvaginally. The incubation period after exposure is 3 to 21 days [12]. Factors that predispose women to this infection include multiple sexual partners, pregnancy, and menopause [9,12].
Is the presence of \textit{Trichomonas} predictor of coinfection

Trichomoniasis in women causes copious amounts of a thin, frothy, green-yellow or gray malodorous vaginal discharge [12]. Other common symptoms include vulvovaginal irritation, vaginal soreness, and dyspareunia [12]. Only approximately 25% of women with TV infection will be asymptomatic [9].

On physical examination, copious amounts of vaginal discharge is often present. A strawberry cervix or redness of the vagina or perineum has also been described [12].

The diagnosis is frequently confirmed by the presence of motile trichomonads observed on a wet mount preparation of vaginal secretions. However, there are many false-negative results associated with the use of the wet mount preparation as the definitive test [12,13]. Reported sensitivity of the wet mount preparation for \textit{Trichomonas} ranges from 52% to 93% [14,15] The gold standard has traditionally been considered broth culture [15,16]. Recently, several new tests, each with varying sensitivities, have become available for use, including polymerase chain reaction (PCR) technique with a 95% sensitivity, enzyme-linked immunosorbent assay with 82% sensitivity, and direct fluorescence antibody testing with 85% sensitivity [16].

Both urine and vaginal specimen PCR tests have been developed, with PCR vaginal specimen testing having a greater sensitivity than urine (89% vs 64%) [17].

\textit{T vaginalis} has been regarded by some as a useful marker for the presence of other asymptomatic sexually transmitted infections such as NG and CT in women. In one such study, researchers examined the presence of other sexually transmitted infections in women with TV during 2 periods, 10 years apart, at the same institution. For 1983, they found 20% of women infected with TV were also infected with NG; for CT the percentage was 15% of TV-infected women [8]. For 1993, they found approximately 30% of women with TV had at least one other STD. For TV-positive women, the prevalence of NG was 10% (vs 20% 10 years prior). For CT, the prevalence of coinfection was unchanged at 15% in TV-positive women. The authors of this study concluded that TV was still frequently associated with other pathogens and screening of these women for other infections was essential [8].

In our study, we found a nearly identical prevalence of NG in our TV-positive female patients (9.8% vs 10%) compared with the study by Reynolds and Wilson, but only half the prevalence of CT (8.3% vs 15%). The Reynolds study is limited however in that they did not have a control group (ie, TV-negative) as in our study.

Emergency physicians are frequently required to treat female patients with suspected NG and/or CT infection based on clinical suspicion alone because the results of the tests commonly used for confirmation of these organisms are not available until after the ED visit. Unfortunately, EPs do not always recognize the presence of these infections. In one retrospective study of female patients presenting to the ED with NG and/or CT, only 45% of patients testing positive for these organisms were recognized and appropriately treated in the ED [5]. Unfortunately, approximately 9% of the patients not initially treated in the ED never received treatment. The median time for treatment for those patients that did follow-up was 36 days [5]. A similar study examining the prevalence of NG and CT in the ED population (both male and female) found that only 24% of infected individuals were recognized and treated on their initial ED visit [2]. Finally, in a study by Yealy and colleagues [4], they found that only 53% of female patients with positive cultures for NG or CT were treated before ED release. For 25% of the patients not treated, no follow-up occurred because of inability to locate the patient. An additional 20% of women did not receive treatment for 14 to 60 days [4]. Clearly, a timely marker is needed that could improve the EP’s suspicion for the presence of CT and/or NG in patients presenting to the ED with symptoms consistent with an STD.

The wet mount preparation is commonly used for the detection of TV. Although the test is not 100% sensitive, it does have the distinct advantage of a rapid turnaround, with the results readily available to the EP to make treatment decisions. If the presence of TV was found to be a reliable marker of coinfection with CT and/or NG, this would offer 2 distinct advantages to the EP. First, EPs could be more confident in their decision to empirically treat for NG and CT in TV-positive female patients suspected of harboring an STD. Secondly, the presence of TV might raise the suspicion of other STDs in patients for which NG and CT infection was not initially considered by the EP and result in appropriate treatment.

From the results of our study however, the presence of TV cannot be considered a reliable marker for the presence of coinfection with NG and/or CT. Emergency physicians will need to continue the current practice of empirically treating high-risk patients and calling back the non–high-risk patients with positive test results for treatment or appropriate follow-up.

There are several limitations to our study. First, like all retrospective studies, there was a significant number of patients, both TV-positive and TV-negative, for which the results of the CT and NG testing were not available. This limitation however affected both the study and the control group. Secondly, we used the wet mount preparation for the testing of TV rather than the gold standard culture. We did this for 2 reasons. First, the wet mount preparation is the more common technique used in EDs to detect the presence of TV. Secondly, the point of the study was to have an early marker for the presence of coinfection with NG and CT; a culture would not serve as such an early marker. It might be worth repeating this study with one of the newly developed techniques, such as PCR. Finally, we did not attempt to correlate the clinical presentation of our patients with their test results. It was not the purpose of our study. We feel confident that an STD was suspected in all 690 patients based on the simple fact that every patient in our study underwent testing for TV, bacterial vaginosis, NG, and CT.
We found, as others have, that patients with TV infection are often infected with NG and CT; however, there is no statistically significant increase in the prevalence of these organisms from TV-negative female patients. The presence of TV cannot be used as a reliable marker of coinfection with NG or CT in female patients presenting to the ED with symptoms consistent with an STD.

References

Use of an emergency department by nonurgent patients

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Received 7 August 2003; revised 28 April 2004; accepted 10 May 2004


Abstract

Objectives: The objectives of this study are (1) to determine whether patients seeking emergency department (ED) nonurgent care have primary care providers (PCP) or know of other care sources and (2) to determine the reasons why they choose to use the ED.

Methods: A cross-sectional survey in a university ED was administered to self-referred nonurgent patients for 6 weeks. Use of a PCP, knowledge and attempts to seek other care, past use of the ED, urgency self-report, time of visit, and reasons for choosing an ED were recorded.

Results: Of the 563 approached subjects, 314 were eligible and 279 agreed to participate. One hundred fifty-seven (56%) had PCPs. For 183 (66%) subjects, the ED was the only place they knew to go for their present problem, and 75 (27%) reported that they depended on the ED for all medical care. Of those patients with a PCP, 73 (47%) rated the ED better for unscheduled care. Eighty-one (52%) subjects thought their PCP would be more efficient and 66 (42%) thought their PCP would be cheaper.

Conclusions: Although most ED nonurgent patients were not dependent upon the ED, the majority was unaware of other places to go for their current health problem. Even those patients with a PCP sought care in the ED because the ED was believed to provide better care despite its perceived increase in both waiting time and cost.

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1. Introduction

There has been a steady increase in the use of emergency departments (ED) since the mid 1940s [1]. Some might dismiss this fact as obvious, citing the increase in visits as a result of overall population growth. However, these increases in ED use have been much more drastic than the increase in population [1-3]. Currently, it is estimated that...
patient visits to an ED for nonurgent complaints are between 33% and 50% [1,2,4-6]. The increased patient load may cause emergency providers to allot less time to care for each patient and to decrease time for health teaching, counseling, and helping patients with chronic health problems to find appropriate resources to help manage their disease states [1]. Given that previous reports found that patients presenting to the ED report longer waits, more costly care, and poor follow-up care [6], the question remains why patients decide to use the ED for nonurgent complaints. Furthermore, as EDs around the country face “overcrowding” issues, what should be done with this increasing nonurgent population?

The purpose of this study is to further investigate the use of the ED by self-referred nonurgent patients (defined as those low-acuity patients that meet Emergency Severity Index [ESI] triage categories of 4 or 5), including (1) whether nonurgent patients presenting to an ED have primary care providers (PCP), (2) what reasons factor into the decision to come to an ED for a nonurgent complaint, (3) if nonurgent patients know of other sources of care for their complaints, (4) if there is a difference in regards to the previous 3 aspects when comparing the patient population that presents between 9:00 AM and 5:00 PM (normal business hours) to those patients who present between 5:00 PM and 1:00 AM, and (5) if these nonurgent patients have insurance.

2. Methods

2.1. Study design and population sample

Using a cross-sectional study design, a convenient sample of patients presenting to the University of North Carolina ED who were triaged as nonurgent by trained nursing staff were interviewed by trained research assistants. Patients triaged to the acute ED and minor trauma areas as nonurgent between 9:00 AM and 1:00 AM, the hours for which research assistants were available, during the study period of June 23, 1999, to August 8, 1999, were invited to participate. Exclusion criteria included those persons who were intoxicated, pregnant, mentally impaired, younger than 18 years, a contract case (those cases that the ED had a contractual obligation to see, such as prisoners or hospital employees), suspected victim of abuse, or those referred by a physician.

2.2. Setting

University of North Carolina Hospital is a tertiary care, level 1 trauma center with an annual patient census of 65,000 triaged patients that serves a suburban/rural population in central North Carolina. The ED is organized into 4 separate areas: (1) acute ED, (2) pediatric ED, (3) minor trauma, and (4) urgent care. The acute ED is the only area open 24 hours, minor trauma is open 12:00 PM to 12:00 AM, the pediatric ED 9:00 AM to 11:00 PM, and urgent care 9:00 AM to 9:00 PM. Any patients who would be triaged to minor trauma, urgent care, or pediatric ED are sent to the acute ED when one of these areas is closed. Board-certified emergency medicine physicians staff the acute ED and minor trauma. The pediatric ED only sees patients 16 years or younger and is staffed by pediatricians. Urgent care is staffed by internal medicine physicians and serves mainly as a walk-in clinic for minor medical complaints. Internal medicine physicians often refer their patients to urgent care when the patient has an acute medical problem and cannot be seen by the physician. Patients who were triaged to the urgent care area were not included in the study population because most of these patients are referred to this area by their PCP or medicine clinic. All patients presenting to the ED are triaged by a trained member of the nursing staff to the appropriate area of care. This study received an expedited approval from the University of North Carolina Committee for the Protection of Human Subjects in Research.

2.3. Triage Scale

Patient urgency was assessed using the ESI triage algorithm, a validated scale on which the nursing staff

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Fig. 1 Questions on the patients’ completed questionnaire.
has received extensive on-going education and evaluation [7-12]. Those patients assigned a triage ranking of 4 or 5 were considered nonurgent. The 4 and 5 ratings are given to those patients who are triaged as very low risk. These patients are responsive, oriented, in no acute distress, have stable vital signs, and estimated to require no more than one resource (lab, test, or consult).

2.4. Survey tool

The survey tool was a 2-part instrument. The principal investigator piloted the tool with actual patients in the ED on 3 separate occasions before the start of the study to refine the survey instrument. The first part of the instrument was a brief patient-completed questionnaire, shown in Fig. 1, which assessed the demographics as well as the degree of

| What problem brought you to the Emergency Department today? |
| Did anyone come with you? If so, who? |
| Where do you usually go for care? |
| Other than an Emergency Department, do you know of any other places you could have gone for this problem today? |
| Did you seek any other sources of care before coming to an Emergency Department today? |
| Today, which of the following influenced your decision to seek care in an Emergency Department: |
| You believed that an Emergency Department: |
| ◆ Will see you quickly and without an appointment (immediacy) |
| ◆ Is always open and allows you to work around your schedule (expediency) |
| ◆ Will bill you later so you do not have to pay today (payment) |
| ◆ Gives better care than any other healthcare options in the area (better care) |
| ◆ Always has a doctor on duty (immediacy) |

If patient answers yes to "Always has a doctor on duty", do the following apply?

◆ You do not have a regular physician.

◆ You do not know of anywhere else to go.

If the patient usually seeks care from a source other than an ED:

◆ How does the Emergency Department compare (better, same, or worse) to your other primary source of care in terms of:
  ◆ Waiting time
  ◆ Quality of care
  ◆ Personal attention from staff
  ◆ Cost
  ◆ Ease of being seen without an appointment

Fig. 2 Interview questions asked by the trained research assistant.

Fig. 3 Parameters of each visit recorded by the research assistant.
worry and urgency (as rated by the subject) that led the patient to come to the ED. A brief interview conducted by trained research assistants was then administered to assess the patient’s reasons for choosing to seek care in an ED by asking the questions listed in Fig. 2. The research assistant also recorded the specific parameters of each patient visit as detailed in Fig. 3.

2.5. Research assistant training

Six research assistants were trained during a 1-hour session to conduct the survey. The principal investigator observed each research assistant during 2 actual patient encounters. Both the research assistant and principal investigator scored the encounter separately. Later, the 2 encounters were compared using the principal investigator’s assessment as the gold standard. In all cases, there was 100% concordance between the research assistant and the principal investigator.

2.6. Statistical analysis

All the data were coded and entered into Microsoft Excel (Microsoft, Seattle, Wash) by the principal investigator. A biostatistical consultant using SAS Research Data Management (SAS Institute, Cary, NC) performed the statistical analysis. Categorical data were analyzed using $\chi^2$ and continuous data with a $t$ test.

3. Results

During the study period, 563 patients were evaluated for exclusion criteria. Of these, 284 were excluded for the following criteria: 50 were younger than 18 years, 4 were pregnant, 28 were intoxicated, 46 were mentally impaired, 26 were non–English speaking, 7 were suspected victims of abuse, 85 were referred to the ED by their physician, 35 refused to participate, and 3 left before they had been

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographics of patients triaged 4 or 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients triaged as nonurgent in the acute ED and minor trauma</td>
<td>Excluded patients</td>
</tr>
<tr>
<td>Age</td>
<td>36.1 years (SD ± 17.6)</td>
</tr>
<tr>
<td>Sex</td>
<td>53.3% male</td>
</tr>
<tr>
<td>Race</td>
<td>58.6% Caucasian</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Expanded demographics of included patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary care physician</td>
<td>56.3% Yes</td>
</tr>
<tr>
<td>Chief complaint (those reported over 5%)</td>
<td>36.2% Orthopedic</td>
</tr>
<tr>
<td>Necks/Back pain</td>
<td>10.0%</td>
</tr>
<tr>
<td>Laceration</td>
<td>9.7%</td>
</tr>
<tr>
<td>Minor medical</td>
<td>8.2%</td>
</tr>
<tr>
<td>Motor vehicle crash</td>
<td>7.5%</td>
</tr>
<tr>
<td>Infection</td>
<td>7.5%</td>
</tr>
<tr>
<td>Insurance</td>
<td>35.8% Private</td>
</tr>
<tr>
<td>Uninsured</td>
<td>31.5%</td>
</tr>
<tr>
<td>Medicaid</td>
<td>9.7%</td>
</tr>
<tr>
<td>Medicare</td>
<td>6.1%</td>
</tr>
<tr>
<td>HMO</td>
<td>3.8%</td>
</tr>
<tr>
<td>Other</td>
<td>11.8%</td>
</tr>
<tr>
<td>Wait time for physician</td>
<td>66.2 min (SD ± 49.1)</td>
</tr>
<tr>
<td>Range, 1-330 min</td>
<td></td>
</tr>
<tr>
<td>Total in time in ED</td>
<td>175.6 min (SD ± 106.0)</td>
</tr>
<tr>
<td>Range, 33-877 min</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Survey results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Where do you usually go for care?</td>
<td>Primary care physician 43.7%</td>
</tr>
<tr>
<td>Only ED 27.1%</td>
<td></td>
</tr>
<tr>
<td>Clinic 16.2%</td>
<td></td>
</tr>
<tr>
<td>Other 11.2%</td>
<td></td>
</tr>
<tr>
<td>Health department 1.8%</td>
<td></td>
</tr>
<tr>
<td>Other than an ED, do you know of any other places you could have gone for this problem today?</td>
<td>Nowhere else 65.6%</td>
</tr>
<tr>
<td>Clinic 17.2%</td>
<td></td>
</tr>
<tr>
<td>Other 6.5%</td>
<td></td>
</tr>
<tr>
<td>Primary care physician 5.4%</td>
<td></td>
</tr>
<tr>
<td>Different ED 3.9%</td>
<td></td>
</tr>
<tr>
<td>Health department 1.4%</td>
<td></td>
</tr>
<tr>
<td>Did you seek any other sources of care before coming to an ED today?</td>
<td>No 74.9%</td>
</tr>
<tr>
<td>Other 14.3%</td>
<td></td>
</tr>
<tr>
<td>Clinic 6.1%</td>
<td></td>
</tr>
<tr>
<td>Primary care physician 3.6%</td>
<td></td>
</tr>
<tr>
<td>Health department 0.4%</td>
<td></td>
</tr>
<tr>
<td>Different ED 0.4%</td>
<td></td>
</tr>
<tr>
<td>On a scale of 1 to 10, how urgent is your condition today?</td>
<td>Median 5.0</td>
</tr>
<tr>
<td>Mean 5.6</td>
<td></td>
</tr>
<tr>
<td>On a scale of 1 to 10, how worried are you about your condition today?</td>
<td>Median 5.5</td>
</tr>
<tr>
<td>Mean 5.7</td>
<td></td>
</tr>
<tr>
<td>Why did you choose the ED as your source of care today?</td>
<td>Better care 76.1%</td>
</tr>
<tr>
<td>Urgency 73.6%</td>
<td></td>
</tr>
<tr>
<td>Immediacy 68.6%</td>
<td></td>
</tr>
<tr>
<td>Payment flexibility 41.9%</td>
<td></td>
</tr>
<tr>
<td>Expediency 39.7%</td>
<td></td>
</tr>
</tbody>
</table>
discharged. There was no significant difference between excluded and included patients in regards to age or sex.

3.1. Patient demographics

Two hundred seventy-nine patients completed the survey and interview. Patient demographics are recorded in Table 1. No significant difference existed between the included patients and the overall population of the ED who received a triage rating of 4 or 5 in regards to age, sex, or race. Expanded demographics for patients included in the study are listed in Table 2.

4. Survey results

The survey results, shown in Tables 3 and 4, reveal that the majority (56.3%) of the questioned population did indeed have a PCP. However, of this majority, only 43.7% received their usual care from a PCP, and 27% were reliant on an ED for all their medical needs.

Furthermore, 74.9% of patients came directly to the ED for care, with 65.6% knowing of no where else that they could seek appropriate care for their complaint at that given time. When queried as to why they decided to seek care in an ED, 76.1% believed that they would receive better care and 73.6% cited that their complaint was urgent.

Most of the population surveyed (68.5%) had some form of insurance, with most (35.8%) possessing private insurance. When comparing the population that came to the ED from 9:00 AM to 5:00 PM (normal physician office hours) to the group that entered from 5:00 PM to 1:00 AM, there was no significant difference in regards to (1) having a primary care physician, (2) whether they sought care/advice before proceeding to an ED, (3) reason for seeking care in an ED, or (4) insurance status.

When asked how worried and how urgent they felt their complaint is, the means were 5.7 and 5.6 on a 10-point scale. Finally, when asked to compare the ED with other sources of care they had encountered in the past, this population felt that an ED is better for obtaining unscheduled care, whereas other sources were better for overall waiting time and overall cost of the visit.

5. Discussion

5.1. Do nonurgent patients presenting to the ED have PCPs?

This study found that most nonurgent patients who use the ED for care do, in fact, have a PCP. However, if most of these patients have a PCP, the question arises as to why they decide to seek out the ED rather than their PCP. It is understandable why those who admitted that the ED is their only source of care came to the ED; it serves the purpose of a PCP. Yet, a large proportion of the patients who stated that they would normally seek out their PCP for medical problems, on these occasions, have ended up in an ED. Other studies have reported similar findings, noting that roughly half (48%-61%) of the nonurgent patients presenting to an ED have a PCP [13,14]. However, reports looking at the ED population as a whole (both urgent and nonurgent) have varied results ranging from 39% to 69% of patients having a PCP [5,13,14].

5.2. What reasons factor into the decision to come to an ED for a nonurgent complaint?

When prompted as to why they chose to come to an ED for care, the 3 most common reasons were (1) belief that they could receive better care at an ED, (2) urgency of their complaint, and (3) immediacy. Patients citing better care included those who did not believe that other sources of care would have the resources needed to properly care for their complaint (eg, a patient who fell and hurt his arm and feared that he broke it, but knew that his PCP did not have an x-ray machine at his office comes because the ED has the radiographic capabilities). Patients citing urgency, a somewhat expected response, believed that their conditions warranted prompt medical attention and feared that delaying care would lead to an adverse outcome. Related to the feeling of urgency is immediacy, the belief that the ED would see patients quickly and without an appointment.

If patients believe that an ED would provide better care, it is understandable why they would come directly to the ED, regardless of the availability of other sources of care. Similarly, patients who feel they are in need of care urgently and/or immediately may also come directly to an ED instead of other sources, because they know that an ED will have adequate sources to treat their complaint—whether it be the ability to run diagnostic tests or obtain specialty consults in a timely fashion.

Although mentioned significantly less frequently, patients also cited payment flexibility and expediency as contributing to the decision to seek care in an ED. The mention of payment flexibility reflects the population’s
knowledge that most EDs do not make patients pay at the time of care, but instead will send bills later. This allows the uninsured who may not have money to seek care from a PCP the opportunity to receive prompt medical attention without the concern of paying at the time of care, placing the ED in the position of a safety net for healthcare for the uninsured. Expediency refers to the fact that an ED may be more convenient for the patient, whether it be because the ED is open 24 hours or the patient has a hectic schedule and has problems making and keeping scheduled appointments with a PCP.

Examining other studies that have surveyed the reasons that nonurgent patients present to an ED most have found similar answers. Habenstreit [5] postulates that those patients who lack PCPs and rely exclusively on the ED for care do so because of early socialization, habit, and convenience. Although it may be true that many of these individuals do not exercise selective judgment when deciding to present to an ED with a nonurgent complaint, other studies have found that nonurgent patients cite the following reasons for their choice of care: (1) high acuity of current ailment [1,13], (2) immediate care available in an ED [1,3,4,13,15,16], (3) easy accessibility [1,3,5,16], (4) quality of care received in an ED superior to other sources [1,5,6], (5) inability to get an appointment with PCP [4,6,16], (6) unfamiliarity with other sources of care [6,16], (7) knowledge that they would not be billed at time of visit [5,16], and (8) overall convenience [1,5].

5.3. Do nonurgent patients know of other sources of care for their complaints?

Most patients in this study did not know of anywhere else to go for their problem on that occasion. However, only 27.1% were completely dependent upon the ED as their sole provider of healthcare. Thus, most patients had received care from a health provider outside an ED and may be expected to know of another source of care besides the ED, although that source may not be appropriate for their immediate healthcare needs.

It has been suggested that many nonurgent patients use an ED as a backup for care, when their PCP is unavailable [5]. But between 9:00 AM and 5:00 PM, the same hours that a typical PCP’s office would be open, an ED sees its greatest number of nonurgent patients [1,17]. It is possible that patients underestimate the level of care that a PCP can provide (or similarly, they overestimate the severity of their complaint). By definition, patients who were triaged as nonurgent were nonacute cases that should have fallen within the scope of a PCP’s care [8]. However, if the PCP does not have the proper resources (radiograph, expedient laboratory results, etc) or time to see the patient, the outpatient PCP system cannot provide adequate care for nonurgent patients that seek immediate care.

Finally, nonurgent patients may seek out care in the ED because they truly believe that their case is an emergency. Patients do not have the clinical experience to accurately assess the severity of their medical complaint; this is complicated by the fact that a problem often seems much more urgent when it is happening to one’s self. There is no way to effectively encourage this group of patients to seek care from other sources.

5.4. Is there a difference in regards to (1) having a PCP, (2) the reasons factoring into the decision to seek care in an ED for a nonurgent complaint, and (3) knowing of other sources of care, when comparing the patient population that presents between 9:00 AM and 5:00 PM (normal business hours) to those patients who present between 5:00 PM and 1:00 AM?

This study found no differences among the population that presented between 9:00 AM and 5:00 PM and that which entered the ED from 5:00 PM to 1:00 AM in regards to (1) having a PCP, (2) the reasons factoring into the decision to seek care in an ED for a nonurgent complaint, and (3) knowing of other sources of care. It was originally conjectured that there would be a difference in these 2 populations in regards to having a PCP—the population that came during 9:00 AM to 5:00 PM was expected to be less likely to have a PCP than the population that sought care from 5:00 PM to 1:00 AM, hours when a PCP would normally be unavailable. This postulation stemmed from the assumption that those patients who had a PCP would seek care from that provider when available (9:00 AM to 5:00 PM), and would come to an ED when the PCP’s office was closed and unavailable. Those patients without a PCP, on the other hand, would be just as likely to seek care from an ED at all hours. This study did not support that conjecture.

5.5. Do these nonurgent patients have insurance?

More than two thirds of the study population had some form of insurance. Previous studies examining the nonurgent population presenting to the ED have mixed findings; some have similar findings, showing that more than 57% of the nonurgent population are insured [16,18], whereas another demonstrated that most of the nonurgent population is uninsured [4]. If most of this population is indeed insured, perhaps it is in the best interest (from a revenue standpoint) for the ED to see and treat these patients.

5.6. Impact of nonurgent patients on the ED

In much of the previous literature examining ED overcrowding, a common proposed solution has been to eliminate nonurgent patients from the ED. However, as the face of the nation’s healthcare system changes, these patients will continue to seek care in the ED until they are denied of care or until they are unwilling to endure the long waits and overcrowded conditions that currently exist. Another poten-
6. Limitations

This study had limitations. Despite attempting to encounter all nonurgent patients who presented to the ED during the study period, only a little over a third of all nonurgent patients were approached to be included. This percentage is due to the time constraints on the research assistant, the high number of nonurgent patients, and the quick turn around time on certain patients. However, the sample group that was surveyed was demographically similar to the whole population that entered the ED during the study period. Also, the ED at University of North Carolina is set up in such a way that the main triage desk serves the ED and the urgent care clinic. The urgent care clinic is run by the internal medicine service and treats minor medical problems—almost all of the patients seen there are triaged as nonurgent. However, many internal medicine physicians tell their patients that if they have problems and cannot reach them, they should go to the urgent care clinic for treatment. In this way, the urgent care clinic operates much like a walk-in medical clinic except that some nonurgent patients thinking they will be seen in the ED may be triaged to the urgent care clinic. Finally, one of the shortcomings of the triage system was that some of the minor trauma cases (particularly lacerations) that were accurately triaged as nonurgent were serious that they may have exceeded the limitations of some PCPs.

7. Conclusion

Among the study population, most nonurgent patients are insured and do not depend upon the ED for routine care. In fact, most have a PCP and would usually consult him/her for medical care. Most of these nonurgent patients, however, are unaware of other sources of adequate care for their current healthcare needs and thus seek out the ED. These patients also use the ED for their nonurgent complaints because they believe that the ED will provide better care in an immediate fashion for what they believe to be urgent complaints.

References

The duration of ventricular fibrillation required to produce pulseless electrical activity

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Received 5 February 2004; accepted 5 March 2004

Abstract The duration of untreated (no cardiopulmonary resuscitation) ventricular fibrillation (VF) needed to produce postdefibrillation pulseless electrical activity (PEA) was determined in 9 anesthetized swine ranging in weight from 20 to 30 kg. VF was induced electrically by a right ventricular catheter electrode, while arterial pressure and the electrocardiogram were recorded. VF was confirmed by the presence of VF waves in the electrocardiogram and a loss of pulsatile arterial pressure. VF was allowed to persist for 15-second increments (eg, 15, 30, 45, etc), after which defibrillation was achieved with transchest electrodes and the presence or absence of PEA was noted. If PEA was present, rhythmic chest compressions were applied to rescue the animal. Just after initiation of VF and just before defibrillation, VF wave frequency was measured. PEA was encountered in 100% of the trials after 180 seconds of VF. The threshold duration for PEA was 60 seconds. VF wave frequency decreased with the passage of time. At VF initiation, VF wave frequency \((f_0)\) ranged from 6 to 15 per second, with a mean of 10.1 ± 2.1 per second. At 180 seconds \((f_{180})\), the mean frequency was 4.0 ± 0 per second. It was only possible to eliminate PEA and restore pumping in 1 animal when untreated VF lasted more than 180 seconds. There was no clear transition in the frequency of the VF waves with the passage of time that could predict the possibility of postdefibrillation PEA. Moreover, because of the different initial VF wave frequencies and the different rates of decrease with time, a measurement of VF wave frequency is unlikely to be informative on how long VF had been present.

A consistent finding in this swine study of prolonged untreated VF was a rise in blood K\(^+\) which increased from a normal prefibrillation value of about 4 mEq/L to 8 to 12 mEq/L at 180 seconds. The longer the duration of VF, the higher the K\(^+\).

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1. Introduction

Pulseless electrical activity (PEA), formerly called electromechanical dissociation, is a postventricular defibrillation condition in which the R waves of the electrocardiogram (ECG) are present but are not followed by ventricular contractions. It likely underlies the reason for the poor outcome of out-of-hospital resuscitation. Identification of PEA requires an ECG and some means to identify the absence of a pulse. No existing automatic external defibrillator (AED) (or implanted cardioverter defibrillator...
Pulseless electrical activity

[ICD]) is equipped with a pulse detector and unless the rescuer views the ECG and palpates the pulse, PEA will be missed and deterioration and death are likely. If the rescuer identifies R waves in the ECG but no pulse is palpable, prompt cardiopulmonary resuscitation (CPR) and advanced life support are mandatory.

The amount of time that ventricular fibrillation (VF) must last, without CPR support, to encounter PEA is, at present, not well documented. The present animal study was undertaken to identify the duration of unsupported VF needed to encounter postdefibrillation PEA.

2. Methods and materials

All studies were performed on 9 anesthetized pigs ranging in weight from 20 to 30 kg. Each animal was sedated, intubated, and anesthetized with isoflurane and oxygen to a depth of Guedel stage 3, plane 2. Femoral artery pressure and lead II ECG were recorded. VF was induced electrically with a right ventricular catheter electrode to which 2-millisecond pulses at 50 per second with an intensity of 10 V were applied. VF was confirmed by the appearance of fibrillation waves in the ECG and a loss of pulsatile blood pressure. As soon as VF was confirmed, the anesthesia was discontinued and restored after ventricular defibrillation. VF was allowed to persist for increasing 15-second increments (eg, 15, 30, 45, 60, etc). At the end of each increment, the ventricles were defibrillated with transchest electrodes. If no R waves occurred because of S-A node arrest or AV block, the ventricles were paced with the right ventricular catheter electrode used to induce VF. In this way, we could test for PEA in all animals. Cardiopulmonary resuscitation was applied with the chest thumper (Michigan Instruments Inc) to recover the animals. Additional trials to identify PEA were not attempted until the animal’s blood pressure returned to the prefibrillation pressure. All data were entered into an online computer (LABVIEW), whereas recordings were made on a stripchart recorder.

Control blood gases and chemistries (pH, K+, Na+, HCO3-, Cl-, hematocrit, hemoglobin, Sao2 levels) were obtained before the induction of VF and immediately after defibrillation using the chest thumper to provide circulation during PEA. During these episodes blood gases and chemistries were obtained.

VF wave frequency was measured on a second-by-second basis on the animals carried to 180 seconds. This was done to determine if VF wave frequency could be used as a predictor of postdefibrillation PEA.

3. Results

Fig. 1 is a record of the ECG and blood pressure that illustrates postdefibrillation PEA. In this record, there is total AV block with an idioventricular rhythm, and the R waves are not followed by blood pressure pulses (left and right). In the center of the record, rhythmic chest compressions were applied between START and STOP; note the blood pressure pulses. On cessation of chest compression, PEA returned.

Fig. 2A is a histogram showing the number of PEA occurrences versus the duration of VF using class intervals of 25 seconds. Fig. 2B shows the same data expressed as the percentage of PEA occurrences versus the duration of VF: this figure was made by summing the data in the histogram.
shown in Fig. 2A. Note that by 80 seconds, there was an 18% incidence of PEA. By 180 seconds, the incidence of PEA was 100%.

Before the induction of VF, the blood gases and chemistries were all normal. In those animals in which PEA was present after defibrillation, the chest thumper was applied, and atrial blood samples were drawn immediately. Because the animal was mechanically ventilated with oxygen, the $P_{O_2}$ and $S_aO_2$ were high. With a mean arterial pressure of 20 to 25 mm Hg provided by the thumper, the pH initially increased because of hyperventilation, then decreased because of supervening metabolic acidosis. The most striking feature was a continued rise in $K^+$ from a normal value of 4 mEq/L to 8 to 12 mEq/L, the magnitude of the increase being dependent on the duration of VF.

The frequency of the VF waves was measured, and shown in Fig. 3 is a typical example, showing the variation in frequency and a gradual decrease in mean frequency with the passage of time. Typically, the largest decrease in VF wave frequency occurred in the first minute. Fig. 4 summarizes the mean VF wave frequency versus the duration of VF out to 180 seconds. We were able to recover cardiac pumping in 1 pig with PEA when VF lasted for 180 seconds. Fig. 4 also shows the percent of postdefibrillation PEA versus the duration of VF using the data from Fig. 2B. By 60 seconds (the threshold for PEA), the mean VF wave frequency was 65% of the initial frequency.

4. Discussion

Decoupling of excitation and contraction in human hearts appears to have been reported first by Dorra [1,2] and Dorra et al [3] who described it in the atria after cardiversion with 250- to 300-J shocks. He called the phenomenon “dissociation electromechaniques,” that is, electromechanical dissociation. The modern term is PEA, and it can occur in the atria and ventricles; however, PEA is usually described in association with failure of the ventricles to contract after an R wave in the ECG.

In the present study, it was a surprise to encounter 100% PEA incidence after 180 seconds of untreated VF (no CPR). It is of interest to note in Fig. 3 the manner in which the VF wave frequency decreased with time. The threshold duration...
of VF for PEA was 60 seconds. However, there is no sharp change in VF frequency at this time. The mean frequency at the onset of VF ($f_0$) was $10.1 \pm 2.1$ per second; at 180 seconds ($f_{180}$), it was $4.0 \pm 0$ per second. The mean ratio ($f_{180}/f_0$) is $0.35 \pm 0.02$. In all cases, the VF wave frequency decreased with the passage of time. The wide range of initial frequencies ($f_0$) and the manner of decrease in each animal make it impossible to use VF wave frequency as an indicator of how long VF was present. The progressive decrease in frequency is an indicator of the increasing difficulty of the myocardium to sustainVF caused by hypoxia. However, an increase in VF frequency during CPR may well indicate the effectiveness of myocardial oxygenation.

In a 29-dog study, Vincent et al [4] induced VF and allowed it to persist from 30 to 180 seconds, then defibrillated to identify the time when postdefibrillation PEA appeared. They found that after 120 seconds of VF, postdefibrillation PEA could be produced reliably. In the present pig study, it required 180 seconds of untreated VF to encounter postdefibrillation PEA in 100% of the trials. However, in a few instances, it was possible to produce PEA after 75 to 100 seconds of fibrillation.

The dominant frequency component in a power-frequency spectrum analysis of VF waves from 41 patients was reported by Stewart et al [5]. Data obtained in short-time segments showed that the dominant frequency decreased with the passage of time. At 3 seconds, the dominant frequency was 5.8 per second, and at 20 seconds, it was 2 per second. A plot of the frequency versus time data resembled a decaying exponential curve.

Power-spectra studies of the ECGs of dogs during VF were reported by Martin et al, [6] the objective being to identify any changes with the passage of time during VF. In the first few seconds of VF, the spectrum exhibited a narrow peak between 15 and 18 per second. In the following 40 seconds, the frequency increased, after which the frequency became low and irregular, losing its characteristics after 60 seconds. Except for the slight rise in frequency, the change is in general agreement with the instantaneous frequency data obtained in the present study.

In another study by the same authors to study electromechanical dissociation (PEA) using dogs, defibrillation was performed after successive periods of VF. In all dogs, PEA was observed after 90 seconds of VF. In the present swine study, 60 seconds was about the threshold for encountering PEA.

Using the Fast-Fourier Transform to analyze the ECG during VF in anesthetized dogs, Carlisle et al [7] reported that the dominant VF frequency with body-surface electrodes, recording from nonischemic hearts, was initially $9.9 \pm 0.7$ Hz, remained above 9 Hz for 70 seconds and then rapidly fell to 5 Hz.

The same authors measured the dominant frequency of VF waves, induced by acute coronary occlusion, to be initially $12.3 \pm 0.2$ Hz. The initial frequency is in agreement with the pig data in the present study.

A consistent finding in the present study was the association of PEA with a high K+ in the blood of all animals. That high potassium is a myocardial depressant has been known since 1883 when Ringer [9] showed that excess Ca++ arrested the heart in diastole. He also found that a high Ca++ arrested the heart in systole. In a series of carefully controlled experiments in which he varied the K+/Ca++ ratio, he found a combination that maintained cardiac function, thus was born Ringer solution.

In a study with human hearts, Singh et al [8] found that with 7 minutes of anoxia, K+ leaked out of myocardial cells.

The high values of K+ associated with prolonged untreated VF, we believe, are caused by the lack of tissue perfusion which impairs the ability of the metabolically driven membrane Na+/K+ ion pump. In the present study, postdefibrillation K+ rose as high as 8 to 12 mEq/L in animals in which VF was prolonged, indicating poor tissue perfusion because the SaO2 during postdefibrillation was 98% and the mean blood pressure was 25 mm Hg with CPR and PEA present. Clearly, poor tissue perfusion with oxygenated blood did not eliminate PEA in those animals that sustained VF for long periods. In other words, poor perfusion with oxygenated blood results in hyperkalemia.

Acknowledgment

The authors thank Melissa Bible and Amy Peterson who were responsible for the anesthesia.

References

[9] Ringer S. A further contribution regarding the influences of the different constituents of blood on the contraction of the heart. J Physiol (London) 1883;4:29-42.
Incidence and severity of recovery agitation after ketamine sedation in young adults

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Received 29 March 2004; accepted 21 April 2004

Abstract

**Purposes:** Psychic recovery reactions after ketamine administration are not uncommon in adults, but yet are rare in children 15 years old and younger. The nature of such reactions has not been previously described in young adults, and accordingly we wished to quantify the incidence and severity of recovery agitation after ketamine sedation in patients aged 16 to 21 years.

**Basic Procedures:** We prospectively collected data on 26 young adults aged 16 to 21 years who received ketamine for emergency department procedures, and treating physicians rated recovery agitation, crying, and unpleasant hallucinations or nightmares each on a 100-mm visual analog scale (0 mm = “none,” 100 mm = “worst possible”).

**Main Findings:** Treating physicians rated agitation and crying as entirely absent (rating 0 mm) in 25 of the 26 patients, and unpleasant hallucinations or nightmares as entirely absent (0 mm) in all 26. The single occurrences each of agitation (rating 46 mm) and crying (rating 23 mm) were not severe and resolved spontaneously without treatment.

**Principal Conclusions:** In this small sample of young adults we observed no serious psychic recovery reactions, mirroring the low incidence of such responses well documented with children 15 years old and younger. This supports the expansion of ketamine use to young adults aged 16 to 21 years.

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1. Introduction

Ketamine has been established as a safe and effective sedative for painful pediatric procedures in the ED [1,2]. This dissociative agent can induce dreaming and hallucinations during recovery, and occasional unpleasant reactions and nightmares have traditionally limited its use in adults [3-6]. Children 15 years old and younger, however, are much less prone to such unpleasant recovery reactions and display milder reactions when they occur [1-3,7,8]. The incidence of recovery reactions in patients aged 16 to 21 years has not been previously described, and thus it is unknown whether such young adults possess a predisposition to such reactions similar to that of children or that of adults.

Encouraged by the minimal incidence of psychic recovery reactions observed in children aged 15 years and younger,
emergency physicians in our ED have been occasionally administering ketamine to young adults. We wished to quantify the incidence and severity of recovery agitation after ketamine sedation in patients aged 16 to 21 years.

2. Methods

2.1. Study design

This prospective case series was approved by our hospital’s institutional review board with verbal consent.

2.2. Study setting and population

We prospectively collected data on a sample of young adults aged 16 to 21 years selected for ketamine sedation in accordance with current standard practice at our suburban university medical center and integrated children’s hospital (ED volume, 55,000 per year). There were no exclusion criteria except those standard for ketamine [1-3].

2.3. Study protocol

Ketamine was administered in accordance with our standard ED protocol. Physicians could coadminister atropine at their discretion; however, it is not our departmental practice to coadminister benzodiazepines [7,8]. We typically provide opioids to ED patients with painful conditions at the earliest opportunity, and thus most patients have received morphine or fentanyl before ketamine is administered.

2.4. Measurements

Upon completion of each sedation the treating emergency physician completed a data form detailing demographic information, American Society of Anesthesiologists physical status, the procedural indication, the ketamine loading dose and the nature of subsequent doses, and supplemental atropine dosing (if administered). They were asked to specify whether or not sedation conditions were adequate to facilitate the procedure. Treating physicians then specified the presence or absence of the following adverse effects: “airway complications,” “hypersalivation,” “emesis,” and “other” (with this latter category requiring explanation). They were then asked to grade 3 aspects of the patient’s recovery—“agitation,” “crying,” and “unpleasant hallucinations or nightmares”—each using an unmarked 100-mm visual analog scale (VAS) with “none” marked at the left side and “worst possible” at the right side. There was then a place on the data form for physicians to provide any explanatory comments.

2.5. Data analysis

We used descriptive statistics to analyze the data. Although we initially had hoped to compile a series of 50 subjects, because of a low rate of ongoing enrollment we discontinued the study after 26.

3. Results

Thirteen different emergency physicians enrolled 26 subjects between January 2000 and May 2003. Although there was no systematic attempt to identify missed cases, regular ongoing queries of the ED physician group did not identify any missed cases or patient refusals of consent. Physicians reported to investigators that they were enrolling consecutive cases.

Patient characteristics and ketamine dosing are shown in Table 1. Physicians chose to give ketamine intravenously (IV) in all of these patients. Midazolam was not administered to any subject either during sedation or recovery. Visual analog scale ratings by treating physicians for recovery agitation were zero for all subjects except one. The

<table>
<thead>
<tr>
<th>Table 1 Characteristics of study subjects (N = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y) Mean 17.3; range, 16.0-19.2</td>
</tr>
<tr>
<td>Weight (kg) Mean 73; range, 50-101</td>
</tr>
<tr>
<td>Sex (female/male) 9/17</td>
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<tr>
<td>ASA physical status</td>
</tr>
<tr>
<td>Class 1 24</td>
</tr>
<tr>
<td>Class 2 2</td>
</tr>
<tr>
<td>Procedural indication</td>
</tr>
<tr>
<td>Fracture reduction 13</td>
</tr>
<tr>
<td>Dislocation reduction 4</td>
</tr>
<tr>
<td>Irrigate open fracture 1</td>
</tr>
<tr>
<td>Joint irrigation 1</td>
</tr>
<tr>
<td>Steinmann pin insertion 1</td>
</tr>
<tr>
<td>Incision and drainage of pilonidal cyst 2</td>
</tr>
<tr>
<td>Incision and drainage of facial abscess 1</td>
</tr>
<tr>
<td>Auricular hematoma drainage 1</td>
</tr>
<tr>
<td>Laceration repair 1</td>
</tr>
<tr>
<td>Foreign body removal from ear 1</td>
</tr>
<tr>
<td>Ketamine loading dose (mg/kg IV) Mean 1.3; range, 1.0-1.9</td>
</tr>
<tr>
<td>Atropine coadministered 13a</td>
</tr>
<tr>
<td>Total number of ketamine doses 1 21</td>
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<tr>
<td>2 3</td>
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<tr>
<td>3 2</td>
</tr>
<tr>
<td>Total ketamine dose (mg/kg IV) Mean 1.6; range, 1.0-3.75</td>
</tr>
<tr>
<td>Sedation conditions adequate 26</td>
</tr>
<tr>
<td>Adverse effects during sedation</td>
</tr>
<tr>
<td>Airway complications 0</td>
</tr>
<tr>
<td>Hypersalivation 0</td>
</tr>
<tr>
<td>Emesisb 2</td>
</tr>
<tr>
<td>Otherc 1</td>
</tr>
</tbody>
</table>

ASA indicates American Society of Anesthesiologists.

a The mean atropine dose, when administered, was 0.5 mg (range, 0.4-0.6).
b Both subjects received ondansetron with no further emesis. A third subject reported nausea, which was treated with promethazine, but did not experience emesis.
c One subject experienced urticaria which the treating physician believed because of concurrent fentanyl.
subject experiencing recovery agitation (VAS rating, 46 mm) was an 18 year-old woman undergoing reduction of tibia and fibula fractures. She received a total ketamine dose of 1.54 mg/kg IV. Her treating physician described her as “agitated and confused presedation,” and during recovery she was “restless,” “confused,” and “repetitive,” but without evidence of hallucinations. Her symptoms resolved without treatment and she was calm at the time of discharge.

Visual analog scale ratings for crying were zero for all subjects except one. The subject experiencing recovery crying (VAS rating 23 mm) was a 17 year-old teenaged boy undergoing reduction of fourth and fifth metacarpal fractures. He received a total ketamine dose of 1.67 mg/kg IV. His treating physician described him as having “several episodes of tearfulness” during recovery, with repeated statements of “I love you, mommy.” There were no hallucinations noted. His symptoms resolved without treatment and he was calm at the time of discharge.

Visual analog scale ratings for unpleasant hallucinations or nightmares were zero in all subjects.

4. Discussion

In this small sample of young adults we observed no serious psychic recovery reactions, mirroring the low incidence of such responses well documented with children 15 years old and younger. This supports the expansion of ketamine use to young adults aged 16 to 21 years.

Ketamine-associated hallucinatory recovery phenomena have been reported in up to 30% of adults, and in developed nations these reactions have traditionally limited ketamine use in this population [3-6]. Manifestations of these recovery experiences are highly variable with vivid reports of psychedelic colors, suspension in midair or outer space, floating down a kaleidoscope, rides in spaceships, out of body experiences, or faceless persons walking around the bed. Some patients report the dreams as extremely frightening; others describe them as pleasant, joyful, fascinating, or bizarre [3,5,6]. Traditionally reported risk factors for these hallucinatory reactions include being adult rather than a child, female sex, rapid IV administration, excessive noise or stimulation during recovery, personality disorders, or subjects who normally dream frequently [3,5,6].

Children may dream during ketamine sedation but these occurrences are usually not frightening. Arguably not all pediatric patients are capable of reporting the specific nature of dreams to researchers; however, the low incidence of crying or agitation during recovery supports the benign nature of ketamine in this age group [1-3,7,8]. Unusual dreams or hallucinations, which might prove disturbing to an adult, do not always provoke distress in a “naïve” child with fewer life experiences.

Should pronounced psychic reactions (eg, nightmares, delirium, excitation, physical combative ness) occur after ketamine, they can be consistently and rapidly pacified by titrated benzodiazepines [3,5,6]. Although intramuscular administration of ketamine is common in children, we recommend the presence of IV access in patients 16 to 21 years of age (as with adults) to facilitate prompt benzodiazepine administration as needed for patient comfort and safety [4]. No benefit of prophylactic benzodiazepines has been demonstrated in children 15 years old and younger [7,8], although such use has been traditional in adults [3,5,6].

The primary limitation to this case series is its small size. Unpleasant recovery reactions may still occur, of course; however, their absence in this series suggests that they are not likely to be common. Larger series would better define the incidence and magnitude of such reactions.

Ketamine sedation for children is a daily occurrence in our ED, and we attribute our low enrollment to a limited need for this dissociative agent in this age range. Most young adults are cooperative enough to permit successful procedural sedation using midazolam and fentanyl (our typical agents for adults), and thus ketamine was reserved for highly select cases in which excessive levels and pain and/or anxiety were anticipated.

5. Conclusions

We noted no serious psychic recovery reactions in this case series of young adults receiving ketamine sedation. This supports the use of ketamine in patients aged 16 to 21 years.

References

Injuries to the hand and digits are commonly seen in the emergency department. Lacerations, contusions, puncture wounds, and fractures comprise the bulk of these injuries. A fracture to the dorsum of the distal phalanx can result in a mallet finger deformity. These fractures must be accurately diagnosed with the proper initial treatment begun. There is some disagreement over the best treatment approach and multiple different splints have been described in the literature. Conservative treatment with a finger splint is most commonly effective. We recommend a modified dorsal finger splint for these injuries. We describe a splint to properly treat the fracture, prevent complications, maximize patient comfort during rehabilitation, and prevent mallet finger deformity.

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1. Introduction

A mallet finger results from a traumatic disruption of the extensor tendon at the distal interphalangeal (DIP) joint. This can result from an avulsion fracture of the distal phalanx, a disruption of the extensor insertion, or a tear of the extensor tendon. After the disruption has occurred, there is loss of active extension of the distal phalanx. Flexion is unopposed by extensor forces and, if untreated, the distal phalanx will gradually assume a fixed flexed position.

The mechanism of injury is generally an axial load that causes sudden, forced flexion of the distal phalanx. The patient usually presents acutely with a painful, swollen fingertip that is noticeably tender and swollen at the dorsal DIP joint. The distal phalanx will not extend against resistance and will usually assume a subtly more pronounced flexed position. A radiograph will most commonly reveal an avulsion fracture at the DIP of the dorsal distal phalanx (Photograph 1).

The treatment of acute mallet finger is splinting [1-5]. There have been many creative splints described in the literature [6-12]. The DIP joint is splinted in 5° to 15° of slight hyperextension. No other digit or joint needs to be included in the splinting acutely; however, if a compensatory swan neck deformity occurs, the proximal interphalangeal (PIP) joint will be included. The splint should remain in
place for 8 weeks. It is important that the patient does not flex the DIP joint during this period of rehabilitation for fear of poor healing and further damage to the extensor insertion. The preferred splint is commonly either a commercially available splint (stack splint) or an aluminum-foam ("Zimmer") splint. The aluminum foam splint may be applied to either the dorsal or the volar surface.

Some of these cases may present days and weeks after the acute injury. After the pain and swelling resolves, the patient will more likely notice the dysfunctional joint, and the mallet finger deformity becomes apparent (Photograph 2). The distal phalanx may sublux volarly, and the mallet finger deformity may progress to a compensatory swan-neck deformity (Photograph 3).

All cases initially evaluated in the emergency department (ED) are safest referred for follow-up to a hand or orthopedic surgeon. Surgical pinning may be needed in cases where greater than one third of the DIP joint is involved, the joint is unstable, or in those refractory to splinting.

2. Methods

Our preferred and recommended splint for these injuries is a dorsally placed splint. The aluminum with foam-backed splint is cut to a length of about 4 cm or the length from the distal tip of the nail plate to the distal creases of the PIP joint. A pair of shearing scissors is used to excavate the middle one third of the foam padding (Photograph 4). Each end of the splint is fashioned to a gradually contoured 5° to 15° of hyperextension (Photograph 5). The patient is instructed to hold the injured finger in hyperextension (Photograph 6). The splint is then affixed to the dorsum of the digit by 2 separate tapings (Photograph 7).

3. Discussion

Hand and upper extremity injuries are common in the ED. A review of the National Electronic Injury Surveillance
System shows that the fingers and hand are the most frequent body parts injured at work and treated in hospital EDs [13]. Fractures of the digits are a significant subset of these injuries, and the distal phalanx is the most commonly fractured phalanx [14,15].

A mallet finger fracture must be correctly diagnosed and treated from the ED to prevent the pain and disability of a mallet finger deformity. A careful history, precise physical examination, and accurate radiographic reading will lead to the diagnosis. The initial treatment is essential. A properly fashioned and applied dorsal splint with referral to a hand or orthopedic surgeon will insure that a mallet finger deformity is best prevented.

There is controversy and disagreement over treatment options [16-20]. However, most agree that in a closed rupture or avulsion fracture, if there is no joint dislocation or instability and less than one third of the articular surface is involved, conservative treatment is preferred [21,22]. A typical treatment regime is 8 weeks of continual splinting. Splinting requires patient compliance and can become uncomfortable while leading to its own complications, including maceration and skin necrosis [23]. Several investigators have noted the complications of splinting these injuries including one study with a reported 45% complication rate [24].
There are several specific issues to keep in mind to prevent potential problems or complications of splinting. The degree of hyperextension should not cause pain or skin blanching. Excessive hyperextension must be avoided because of the potential for blistering and maceration of skin over the dorsum of the joint. The splint itself must not put direct, sustained pressure at the DIP joint. The PIP joint should not be included in the splint unless a compensatory swan neck deformity is noted (Photograph 8). Displaced fractures should be reduced before splinting.

The Kleinert modified dorsal finger splint is an excellent choice for these hand-injured patients. It will insure that the digit is positioned properly, maintained in position, and there is minimal exposure to complications including skin breakdown and vascular compromise. The splints are easily fashioned for form fitting hyperextension, and the excavated area greatly reduces the potential for skin maceration at the DIP joint. The patients position their finger in a comfortable hyperextended position that allows ease of affixing the splint. The splints are inexpensive, provide patient comfort, and encourage patient compliance. The ultimate goal is healing with a pain-free fully functioning DIP joint.

References

Canadian Headache Society criteria for the diagnosis of acute migraine headache in the ED—do our patients meet these criteria?

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Received 16 December 2003; accepted 1 March 2004

Abstract

Introduction: We previously reported that many patients who present to the ED with “migraine” headache do not meet the International Headache Society criteria (IHSC) for the diagnosis of acute migraine.

Objective: The aim of the study was to compare the frequency for which ED patients with migraine headache meet the Canadian Headache Society criteria (CHSC) vs the IHSC.

Methods: This was a prospective, observational study, performed at a community ED. Consecutive patients who presented to study authors with a chief complaint of headache were enrolled. Historical/clinical data were collected on a standardized form. Ninety-five percent confidence intervals (95% CIs) were calculated and Fisher exact test was used as appropriate.

Results: One hundred eighty-nine patients were enrolled in this study. Mean age was 38 years. Females comprised 69% of patients. Thirty-seven percent of patients had prior ED visits for headaches. A positive family history of migraines was present in 35% of patients. Diagnostic imaging was previously performed in 44 of the enrollees to evaluate the cause of their headaches. A total of 43 (23%) patients had a prior diagnosis of migraine. Overall CHSC was met in 18% of patients, compared with 15% of patients who met IHSC. Discharge diagnosis of migraine was made in 41% of patients. Of these patients, 33% met CHSC and 28% met IHSC ($P = .30$). For patients with discharge diagnosis of migraine, 33% of females and 36% of males fit CHSC ($P = .53$), whereas 26% and 36% met IHSC ($P = .34$), respectively. For patients with a prior diagnosis of migraine, 32% met CHSC and 26% met IHSC ($P = .24$). Patients with a prior diagnosis of migraine and/or a discharge diagnosis of migraine met CHSC 31% (95% CI, 22%-40%) of the time vs 25% for the IHSC (95% CI, 16%-34%) ($P = .26$). Four patients without a discharge and/or previous diagnosis of migraine met CHSC; 3 met IHSC.
Conclusions: In our study population, only a minority of patients with headache who have prior diagnosis and/or ED diagnosis of migraine headache met CHSC. The utility of CHSC and/or IHSC to standardize ED patients for headache research may be limited.

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1. Introduction

Migraine headaches are an incapacitating disease accounting for 2.8 million physician encounters each year [1]. Osterhaus et al [2] estimated that $2 to 17.2 billion dollars are lost in productivity each year secondary to this ailment. ED visits to alleviate such symptoms are common.

The recognized “gold standard” for the diagnosis of a migraine remains the International Headache Society criteria (IHSC) (Fig. 1). Use of the IHSC in emergency medicine was recently endorsed by the American College of Emergency Physicians when it was suggested that emergency physicians follow the guidelines set forth by the Headache Consortium in 2000 [3]. Use of the IHSC has standardized the diagnosis of migraines; however, its clinical relevance with respect to ED therapy remains questionable. Olesen and Lipton [4] notes that these criteria are not based on scientific literature. Furthermore, a recent study at our institution demonstrated that only a minority of patients discharged by emergency physicians with a previous and/or discharge diagnosis of migraine met the IHSC [5].

In 1997, the Canadian Headache Society published a modified version of the IHSC [6] (Fig. 2). These criteria were created following a needs assessment performed by

<table>
<thead>
<tr>
<th>Migraine without aura</th>
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<tbody>
<tr>
<td>A. At least five attacks fulfilling B to D</td>
</tr>
<tr>
<td>B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)</td>
</tr>
<tr>
<td>C. Headache has at least two of the following characteristics:</td>
</tr>
<tr>
<td>1) Unilateral location</td>
</tr>
<tr>
<td>2) Pulsating quality</td>
</tr>
<tr>
<td>3) Moderate or severe intensity (inhibits or prohibits daily activities)</td>
</tr>
<tr>
<td>4) Aggravated by walking, climbing stairs, or similar routine physical activity</td>
</tr>
<tr>
<td>D. During headache at least one of the two following symptoms occur:</td>
</tr>
<tr>
<td>a) Phonophobia and photophobia</td>
</tr>
<tr>
<td>b) Nausea and/or vomiting</td>
</tr>
<tr>
<td>E. At least one of the following:</td>
</tr>
<tr>
<td>1) History, physical, and neurologic examination do not suggest one of the disorders listed in group 5 to 11*</td>
</tr>
<tr>
<td>2) History and/or physical, and/or neurologic examination do suggest such disorder, but it is ruled out by appropriate investigation</td>
</tr>
<tr>
<td>3) Such disorder is present, but migraine attacks do not occur for the first time in close temporal relation to the disorder</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Migraine with aura</th>
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<tbody>
<tr>
<td>A. At least two attacks fulfilling B</td>
</tr>
<tr>
<td>B. At least three of the following four characteristics:</td>
</tr>
<tr>
<td>1) One or more fully reversible aura symptoms indicating focal cerebral cortical and/or brain stem functions</td>
</tr>
<tr>
<td>2) At least one aura symptom develops gradually over more than four minutes, or two or more symptoms occur in succession</td>
</tr>
<tr>
<td>3) No aura symptom lasts more than 60 minutes; if more than one aura symptom is present, accepted duration is proportionally increased</td>
</tr>
<tr>
<td>4) Headache follows aura with free interval of at least 60 minutes (it may also simultaneously begin with the aura)</td>
</tr>
<tr>
<td>C. At least one of the following:</td>
</tr>
<tr>
<td>1) History, physical, and neurologic examination do not suggest one of the disorders listed in group 5 to 11*</td>
</tr>
<tr>
<td>2) History and/or physical, and/or neurologic examination do suggest such disorder, but it is ruled out by appropriate investigation</td>
</tr>
<tr>
<td>3) Such disorder is present, but migraine attacks do not occur for the first time in close temporal relation to the disorder</td>
</tr>
</tbody>
</table>

* Group 5 to 11 include headache associated with head trauma, vascular and nonvascular disorder, substance or their withdrawal, nonephalic infection, metabolic disorder, and disorders of the cranium, neck, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures

Reference:

Fig. 1 International Headache Society diagnostic criteria.
members of the Canadian Headache Society [6]. Adjustments to the diagnosis of migraine without aura included expanding headache duration, more flexible pulsatile symptoms, and adding osmophobia as a possible diagnostic criterion. In addition to the changes made to the IHSC criteria, they suggest adding additional questions to improve pattern recognition of migraine [6].

We suspected that the Canadian Headache Society criteria (CHSC) might allow for broader standardization of patients with vascular-type headaches who present to the ED. The purpose of this study was to compare the frequency for which ED patients with migraine headache met the CHSC vs the IHSC.

2. Methods

2.1. Study design

This was a prospective observational study.

2.2. Setting

The study was conducted in the Department of Emergency Medicine at Morristown Memorial Hospital, a community-based tertiary care center in northern New Jersey between October 2001 and August 2002. The ED has an annual census of approximately 65,000 visits and has an active academic program and emergency medicine residency. The institutional review board at our institution approved this study before patient enrollment.

2.3. Population

We enrolled a convenience sample of adult patients presenting to the ED with a chief complaint of headache. Each of these patients presented to 1 of 6 emergency physicians. Patients were excluded for any of the following reasons: (1) medically unstable as determined by the attending physician; (2) patient found to be disoriented, intoxicated, and/or with an altered mental status; (3) headache associated with head trauma. In addition, patients were also excluded if they declined permission to participate.

2.4. Study protocol

Informed consent was obtained from participating patients before enrollment. Patients and physicians recorded relevant demographic and clinical variables on a standardized data collection form. Both the IHSC and the CHSC

| Table 1 Characteristics of patients with headache enrolled in the study |
|---------------------|--------|--------|--------|
| No. of patients     | Overall| CHSC   | IHSC   |
| Age (mean, y)       | 38     | 35     | 38     |
| Sex                 |        |        |        |
| Female              | 130 (69%)| 27 (79%)| 22 (78%)|
| Male                | 59 (31%)| 7 (21%) | 6 (22%) |
| Race                |        |        |        |
| Asian               | 8 (4%) | 1 (3%) | 1 (3%) |
| Black               | 25 (13%)| 3 (8%)  | 1 (3%)  |
| Caucasian           | 125 (66%)| 25 (74%)| 21 (75%)|
| Hispanic            | 23 (12%)| 5 (15%) | 5 (18%) |
| Other               | 8 (4%)  | 0      | 0      |
| Previous ED visits  | 70 (37%)| 24 (71%)| 19 (68%)|
| Neurologist following| 45 (24%)| 14 (41%)| 13 (46%)|
| Family history      | 66 (35%)| 18 (53%)| 14 (50%)|
| Previous medications|        |        |        |
| None                | 39 (20%)| 4 (12%) | 2 (7%)  |
| β-Blocker           | 2 (1%)  | 0      | 0      |
| Ergotamine          | 1 (1%)  | 0      | 0      |
| Narcotic            | 23 (12%)| 4 (12%) | 4 (14%) |
| NSAID               | 92 (49%)| 13 (38%)| 11 (40%)|
| Antidepressant      | 6 (3%)  | 2 (6%)  | 2 (7%)  |
| Antiemetic          | 3 (2%)  | 0      | 0      |
| Acetaminophen       | 45 (24%)| 4 (12%) | 3 (11%) |
| Immitrex            | 22 (12%)| 9 (24%) | 7 (25%) |

NSAID indicates nonsteroidal anti-inflammatory drug.
were not identified among the variables addressed on the instrument.

2.5. Statistical analysis

Data were entered into Microsoft Excel for Windows (Microsoft Corporation, Redmund, Wash). Categorical data were analyzed by Fisher exact tests. Ninety-five percent confidence intervals (95% CIs) were calculated as appropriate. All tests were 2-tailed with \( \alpha \) set at .05. The primary outcome parameter was the number of patients diagnosed with migraine upon discharge, and/or who had received a previous diagnosis of migraine, who met either the CHSC or the IHSC.

3. Results

Between October 2001 and August 2002, a total of 189 patients were enrolled. All eligible patients agreed to participate and were surveyed. Mean age of participants was 38 years (\( \pm 13.7 \) SD); females comprised the majority of enrollees (69%) and 66% were Caucasian (Table 1).

With respect to historical features, 70 (37%) of patients had previous ED visits for similar headaches. Imaging modalities (ie, head computed tomography scan and/or magnetic resonance imaging) had been performed in 44% of enrollees previously to determine the cause of their headaches. With respect to family history, 35% of patients had a family member with a chronic headache pattern. Physicians had previously provided a diagnosis of the headache as migrainous in 81 (43%) patients.

Only a minority of patients met either the CHSC or the IHSC. Of the total patients enrolled in our study, only 34 (18%) met the CHSC compared with 28 (15%) meeting the IHSC for the diagnosis of migraine. Discharge diagnosis of migraine was made by the caring emergency physician in 41% of patients overall (Fig. 3). Of these patients, no statistical improvement was demonstrated with regard to either criterion. Of those patients discharged with the diagnosis of migraine, 33% (26 of 78) met CHSC and 28% (22 of 78) met IHSC (\( P = .30 \)). For patients with a prior diagnosis of migraine, 32% (26 of 81) fit CHSC and 26% (21 of 81) fulfilled IHSC (\( P = .24 \)). No significant differences existed in relation to sex. Only 33% (21 of 64) of females and 36% (5 of 14) of males met CHSC (\( P = .53 \)), whereas 26% (17 of 64) and 36% (5 of 14) met IHSC (\( P = .34 \)), respectively. Patients with a prior diagnosis of migraine and/or a discharge diagnosis of migraine met CHSC 31% (29 of 95) of the time (95% CI, 22%-40%) vs 25% (24 of 95) (95% CI, 16%-34%) for the IHSC (\( P = .26 \)). Uniformly, no differences occurred with regard to either criteria used. Four patients without a discharge or previous “migraine” diagnosis met CHSC. This also occurred in 3 patients who fulfilled migraine diagnosis using the IHSC.

![Fig. 3](Discharge diagnosis by number of patients)
Analysis of the individual modifications in the CHSC revealed no significant changes from the IHCS. Addition of pulsatile nature or osmophobia did not increase the number of patients who met diagnostic criteria for migraine. Six more patients were diagnosed with migraine, solely based on increase in headache duration.

4. Discussion

Members of the Canadian Headache Society used a multidisciplinary group reviewing literature and comparing alternative clinical pathways to develop their standard definition of migraine headache. Modifications of the IHSC were intended on improving the reliability of interpretation of the individual diagnostic criteria. The group based their recommendations on the most informative, statistically appropriate references that were available at the time [6].

The first modification pertains to duration. The IHSC requires a duration of 4 to 72 hours. The CHSC expands on this to include those headaches from 2 hours up until 72 hours. With this modification alone, there was not statistically significant increase in patients who met a standard definition of migraine; only 6 more patients were included based on this criterion. Second, the CHSC inclusion of osmophobia (fear of odors) as a feature of migraine did not result in any additional patients who could be considered as having a migraine. Overall, osmophobia was found in 29% of all patients with headache presenting to the ED. Likewise, the addition of pulsating quality at any phase of the attack failed to add additional patients to the diagnosis of migraine by CHSC. Further analysis of this symptom demonstrated that no patient diagnosis was included or excluded based on pulsating quality for the IHSC or CHSC.

Overall, our results demonstrate that most patients in our ED population, who were diagnosed with migraine headaches previously and/or in the ED, did not fit the CHSC. These results mirror those of our previous study, in which we found that most patients with migraine evaluated in the ED did not meet the IHSC [5]. In our current study, the modifications of the CHSC did not provide for the opportunity to standardize additional patients as having migraine headaches when compared with the IHSC.

The utility of strict inclusion criteria for migraines has been questioned. In a prospective observational study of migraine patients, Ducharme et al [7] demonstrated that at 24 to 72 hours after discharge, no significant difference occurred in migraine recurrence between those meeting the IHSC and those diagnosed at the discretion of the ED attending. Analysis using the CHSC was not performed, but the results of this study lead us to conclude that the results would be similar.

Thus, the paradox for investigators in the area of ED headache research remains with respect to classification. Attempts to define migraines by rigid criteria such as the IHSC or CHSC will inevitably result in exclusion of the majority of patients who present to the ED with a “benign” headache. On the other hand, failure to standardize patients in headache research will result in inclusion of patients with different etiologies who may respond differently to various therapies. In the study of Vinson et al [8] of 490 benign headaches, it was found that more than 40% of patients were discharged with the diagnosis of unspecified headache. Similarly, 37% of patients in both our previous and present study had the nebulous diagnosis of undifferentiated headache upon discharge [5].

We suggest further modification of the IHSC for the purpose of ED research. For example, criteria for number of previous episodes could be decreased, but not eliminated. It is important to recognize the recurrence of this ailment. Limitations based on duration have minimal clinical relevance. People often have difficulty estimating this, leading to inaccuracy and questionable validity. The expanded duration used by the CHSC seems appropriate. Osmophobia and pulsatile quality should be excluded from diagnostic criteria as both appear to add little to standardizing patients. It is anticipated that these modifications would be as sensitive and less onerous without a significant loss of specificity (Fig. 4). Implementation, along with validation, of these modifications should ultimately be correlated with treatment modalities.

The utility of diagnostic criteria is important, not only for research but also to allow physicians to standardize diagnostic and treatment modalities. Stringent diagnostic criteria theoretically can assist in eliminating other concerning underlying diseases and further eliminate

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A. At least three attacks fulfilling B to D
B. Headache attacks lasting 2-72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following characteristics:
   1) Unilateral location:
   2) Moderate or severe intensity (inhibits or prohibits daily activities)
   3) Aggravated by walking, climbing stairs, or similar routine physical activity
D. During headache at least one of the two following symptoms occurs:
   a) Phonophobia or photophobia
   b) Nausea or vomiting
E. There is no evidence form the patient’s history or physical exam of any other disease that might cause headache.

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Fig. 4 Modified IHSC.
needless diagnostic testing. These, in essence, can be used as a clinical guideline to help streamline physician’s diagnostic modalities, limiting resources, while saving time, and money. All of which is paramount in medicine today. Is it imperative that we make an accurate diagnosis of migraine and that clinical criteria are both validated and used.

5. Limitations and future questions

The major limitation of this study is the potential for selection bias. Enrollment of patients was based on a convenience sample, not a true consecutive series. While on duty, study emergency physician investigators enrolled consecutive patients who were eligible for the study. Work schedules of each varied, including mornings, evenings, and overnight shifts. Therefore, we suspect that our study population sample represented our diverse patient population with headache as a whole.

This study leaves multiple questions for further research and debate in view of our findings. Only a minority of patients fit either the CHSC or the IHSC. Should we continue to use these as guides for future research? What diagnostic criteria should further migraine studies use? Does the use of the IHSC/CHSC, which excludes a large segment of the headache population, render this research, in many ways, less useful? Should further modification of these criteria be considered? What is the treatment modality of choice for “undifferentiated headaches,” and will those headaches thought to be migrainous respond to similar therapeutic modalities of analgesic control?

6. Conclusion

In our study population, less than half of patients with headache who have prior diagnosis and/or ED diagnosis of migraine headache met CHSC. The utility of CHSC and IHSC to standardize ED patients for headache research may be limited. Further modification of the CHSC/IHSC for emergency medicine research should be considered.

References

Validity of simple measurement to diagnose pupillary dilation☆

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Received 5 March 2004; accepted 7 March 2004

Abstract
Study Objective: The aim of the study was to compare the validity of 3 methods of detecting pupillary dilation: bright-light measurement, room-light measurement, and gestalt judgment.

Methods: In each volunteer, by random assignment, placebo was instilled in one eye and dilute phenylephrine in the other. Emergency care providers judged whether each pupil was dilated and measured it in bright light (>54,000 lux) and in room light (2700-5400 lux) while the other eye was covered. Test characteristics for measurement were determined according to published cut-points, and measurement methods were compared using receiver operating curve analysis.

Results: There were 136 pupillary assessments—68 in placebo and 68 in phenylephrine eyes. Compared with gestalt judgment, bright-light measurement had higher specificity (0.94 vs 0.68) but lower sensitivity (0.43 vs 0.79). Bright-light measurement was more discriminating than room-light measurement.

Conclusion: Bright-light measurement has higher specificity, but lower sensitivity, than gestalt judgement, and is superior to room-light measurement.

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1. Introduction

Pupillary dilation occurs in a variety of toxicologic, neurologic, and ophthalmic emergencies. Bilateral pupillary dilation is caused by overdose with either a sympathomimetic or an anticholinergic agent and by increased intracranial pressure [1]. Unilateral pupillary dilation, which may indicate ocular pathology or uncral herniation, is usually diagnosed by anisocoria, but the contralateral pupil is not always a suitable control; prior iridectomy, enucleation, or other pathology can render anisocoria useless. In certain situations, detection of pupillary dilation may indicate a need for high-risk interventions such as intubation, physostigmine therapy, or neurosurgery.

Health care providers are generally capable of detecting obvious pupillary dilation, as with a “blown” pupil, by gestalt. However, to diagnose pupillary dilation only when it is obvious is to miss opportunities for timely intervention. Unfortunately, health care providers have shown poor agreement in diagnosing mild to moderate pupillary dilation by gestalt [2].

Until recently, the range of normal pupillary size has been described only crudely. A recent report described the
range of normal size in 2 light intensities available in clinical settings: “bright” light produced by handheld sources (>54,000 lux) and fluorescent or “room” light (2700-5400 lux) [3]. Pupillary measurement in these light intensities has good interrater reliability [2]. This report compares the validity of 3 methods of diagnosing pupillary dilation—bright-light measurement, room-light measurement, and gestalt judgment.

2. Methods

2.1. Study design

This design featured an experimental component, in which a mydriatic agent was instilled into some pupils, and a cross-sectional component, in which health care providers measured the pupils and judged whether they were dilated. The design was approved by the institutional review board.

2.2. Study setting and population

In an emergency department, volunteers with no baseline anisocoria received mydriatic and placebo, and then health care workers (attending physicians, residents, registered nurses, physicians’ assistants) recorded observations on dilated and nondilated pupils.

2.3. Study protocol

The study protocol has been described in detail previously [2,3], but is summarized here for the reader’s convenience. Each volunteer had a dilute phenylephrine solution instilled in one eye and placebo in the other; the eye with active drug was determined by random, double-blind assignment.

After a 1-hour waiting period, the data collection protocol began. First, the principal investigator had the volunteer arbitrarily cover one eye and measured the exposed pupil in room light and in bright light. Second, he measured the other pupil with the first one covered. Third, he observed the volunteer with both eyes uncovered to check for obvious anisocoria (>2 mm), which was evidence of drug effect.

If a drug effect was evident, additional emergency department personnel recorded observations in similar fashion as the principal investigator. In the first pupil observed, the provider recorded measurements and a gestalt judgement of whether the pupil was dilated. If the same provider also observed the other pupil, the provider recorded only measurements for the second pupil seen. When adequate numbers of providers were available, each observed only one pupil, and a maximum of 2 observers observed each pupil. Thus, in recording a gestalt judgment of pupillary dilation, providers could observe the pupil in bright light and room light but could not observe the contralateral pupil.

Because the data from this report were collected at the same time as data used to define the range of normal pupillary size in different light intensities, the observers in this study did not have knowledge of these reference ranges [3].

2.4. Data analysis

This report analyzes only pupils in which gestalt judgments were taken by observers other than the principal investigator. For all analyses, the gold standard for pupillary dilation was exposure to phenylephrine, and each observation was used as a unit of analysis.

Receiver operating characteristic (ROC) curves for detection of pupillary dilation were created based on measurements taken in room light and bright light. Software from Metz was used to provide maximum likelihood estimates for ROC curve coordinates, area under the curve, and confidence intervals (CIs) for coordinates and area under the curve [4]. Separate ROC curves were estimated from measurements taken by the principal investigator and by other observers.

Test characteristics—sensitivity, specificity, and accuracy—were calculated for gestalt judgments by each type of professional (attending physician, resident, registered nurse, physicians’ assistant) and for the group as a whole. Test characteristics were also calculated for detecting pupillary dilation using a bright-light measurement cut-point of 3.6 mm and a room-light cut-point of 5.0 mm, the upper limits of normal published previously [3]. Likelihood ratios for a positive (LR[+]) test and a negative (LR[−]) test and 95% CIs were calculated as described by Simel et al [5]. Confidence intervals about proportions were calculated using a normal approximation of the binomial distribution.

The ROC curves and test characteristics were used to compare the validity of the different methods to detect pupillary dilation. Bright-light and room-light measurement methods were compared with each other using the area under the curve. Gestalt judgment was compared with measurement by direct comparison of individual test characteristics as calculated above and by comparison of gestalt judgment sensitivity with a point on the ROC curve corresponding to the same specificity.

3. Results

There were 136 complete assessments taken from 75 subjects—68 in phenylephrine-exposed eyes and 68 in control eyes. Sixty of these assessments were taken with 1 observer measuring one eye, and 76 were taken with 2 observers measuring the same eye. The distribution of assessments by providers other than the principal investigator was as follows: attending physicians, 24 eyes; residents, 42 eyes; registered nurses, 62 eyes; physicians’ assistants, 8 eyes. The degree of dilation was moderate, according to measurements by the principal investigator,
with median bright-light measurements of 3.6 vs 2.5 mm and median room-light measurements of 5.0 vs 3.4 mm.

Table 1 details the test characteristics of gestalt judgment for each type of provider and the performance of simple measurement. Providers’ gestalt judgments had similar specificity for each type of provider, though there was a trend toward higher specificity among residents and attending physicians vs higher sensitivity for registered nurses. Compared with gestalt judgment, both types of measurement had higher specificity and LR(+), but lower sensitivity and LR(−) and slightly lower accuracy.

Fig. 1 illustrates the ability of simple measurement, in bright light and room light, and of gestalt judgment, to distinguish dilated from control eyes. Bright-light measurement was superior to room-light measurement for measurements taken both by the principal investigator and others. For measurements by the principal investigator, the area under the curve was 0.9 for bright light vs 0.84 for room light, with a 95% CI for the difference of −0.03 to +0.16. For measurements by others, the area under the curve was 0.79 for bright light vs 0.74 for room light, with a 95% CI for the difference of −0.06 to +0.16.

As the figure also shows, the point for gestalt judgment by others was slightly above the ROC curves for simple measurement by these observers. However, it was below the ROC curves for principal investigator measurements from these same pupils. At a specificity of 0.68, estimated sensitivities were as follows: bright-light measurement by the principal investigator, 0.91 (95% CI, 0.79-0.96); bright-light measurement by others, 0.75 (95% CI, 0.63-0.88); gestalt judgment by observers, 0.79 (95% CI, 0.68-0.88). Thus, at this specificity, gestalt judgment was not substantially more sensitive than bright-light measurement.

### 4. Discussion

The recent description of normal pupillary size in intensities found in clinical settings renders measurement a more precise means of detecting pupillary dilation [3]. This report shows that bright-light measurement is more valid than room-light measurement in detecting pupillary dilation. Bright-light measurement, using the previously described cut-point of 3.6 mm, has higher specificity and lower sensitivity than gestalt judgment. At comparable specificity, however, gestalt judgment and simple measurement have similar sensitivity.

In answering the question, “Is this pupil dilated?,” clinicians may use a variety of factors: the pupil’s size, the ambient light intensity, the amplitude of the light reflex, the speed of the light reflex, the accommodation reflex, and, if known, the prior appearance of the patient’s pupil. In this study, despite this additional information, including familiarity of the observers with the subjects’ prior appearance in many cases, gestalt judgment was no better than simple measurement. In a real-life situation, clinicians may also incorporate other information into
their gestalt judgment, such as a prior belief that a pupil may be dilated or the fear of intervening if the pupil is dilated.

A test’s influence on a clinician’s belief about the presence of disease, the difference between pretest and posttest probability, can be quantified with a likelihood ratio. A test with a LR(+) value of 10 strongly increases the posttest probability of disease, whereas a test with a LR(−) value of 0.1 strongly decreases the posttest probability of disease [6]. Though the LR(+) values in this study were lower, a low spectrum of disease dampens test performance [7]. If there were more obvious dilation, gestalt judgment and measurement would both have had better likelihood ratios. Nevertheless, a pupil size of greater than 3.6 mm in bright light provided stronger evidence for pupillary dilation than did gestalt judgment.

In this study, gestalt judgment tended toward the middle of the ROC curve, rather than the areas of high specificity or sensitivity. This may have reflected the study setting—there was no higher or lower penalty for false positives vs false negatives; observers were asked, in a comfortable situation, to simply indicate whether the pupil was dilated or not. In this setting, observers may have attempted to maximize accuracy or minimize misclassification. If there was a relatively greater penalty for a false positive, as what occurs when a diagnosis of pupillary dilation necessitates high-risk treatment, such as neurosurgical intervention or physostigmine therapy, one desires higher specificity. Here, gestalt judgment should operate at the left side of the ROC curve, and one hopes that, when faced with a high-risk situation, gestalt judgment would tend in this direction. However, if one does after judgment of pupillary dilation based on a clinical situation, this provides an additional reason to measure pupil size rather than rely on gestalt judgment. To the extent that the clinical situation influences gestalt assessment of pupillary dilation, the pupil’s actual appearance has less independent influence on decision making. By contrast, abnormal vital signs, which are not subject to interpretation, have a great ability to influence decision making and frequently give clinicians reason to reconsider their decisions.

The agent used in this study was phenylephrine, which produces mydriasis without cycloplegia [8]. This agent was chosen, rather than an anticholinergic agent, to avoid unduly favoring bright-light measurement over room-light measurement. The presence of cycloplegia, as what occurs with anticholinergic overdose or uncal herniation, would render bright-light measurement a relatively better test because the light reflex would be abolished. Even without cycloplegia, measurement in bright light was still more discriminating than measurement in room light. For this reason, and for the many practical reasons described in an earlier report, measurement in bright light is preferred when seeking to diagnose pupillary dilation [3].

This study had some notable limitations. First, as noted in a previous report, the limited age range precludes generalizing findings to children or to the elderly [3]. Second, because phenylephrine does not produce cycloplegia, the relative merit of gestalt judgment, which may incorporate the amplitude of light reflex, may have been underestimated. Nevertheless, one expects cycloplegia to also benefit bright-light measurement. Third, allowing the principal investigator to select the number of observers for a given pupil may have biased the study toward including more observations of more difficult pupils. Still, because gestalt judgments and measurements were paired, one would not expect a bias in a comparison of the ability of different methods to diagnose pupillary dilation. Fourth, by preventing observers from observing the contralateral pupil, the study protocol eliminated the method usually used to detect unilateral pupillary dilation; our findings are most generalizable to situations involving bilateral pupillary dilation, such as poisoning.

In summary, in the diagnosis of pupillary dilation, bright-light measurement is more discriminating than room-light measurement. Bright-light measurement, using a cut-point of 3.6 mm, has higher specificity and lower sensitivity than gestalt judgment. Bright-light measurement is preferable to gestalt judgment in cases in which pupillary dilation indicates a need for a high-risk intervention.

Acknowledgments

I thank Dr Deepi Goyal for his help with the design of this study and for coauthoring other manuscripts from the project. I thank all the study participants for their trust and donation of time.

References

Orthopedic pitfalls: cauda equina syndrome

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Received 9 March 2004; accepted 9 March 2004

Abstract Low back pain is an extremely common complaint encountered by emergency and primary care physicians. Although the majority of patients have uncomplicated benign presentations, there is a small subset who has a much more severe disease process called cauda equina syndrome, which entails acute compression of the nerve roots of the cauda equina. These patients usually present posttraumatically with the triad of signs and symptoms including insensate buttocks and/or perineal areas (the so-called saddle anesthesia), bowel or bladder dysfunction, and lower extremity weakness. Significant morbidity can result from delayed diagnosis and treatment; therefore, the emergency physician should remain aware of this potential orthopedic pitfall. This case report discusses the clinical presentation, diagnosis, and relevant treatment of cauda equina syndrome in the ED.

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1. Introduction

Low back pain is a common presenting complaint in EDs and primary care clinics. Approximately 90% of adults will experience back pain at some point in their lives [1]. Most patients’ pain will ultimately resolve spontaneously within 1 to 2 months without a definitive etiology of pain and with minimal intervention and a favorable outcome. Multiple causes of back pain must be considered; the patient’s age, current and past medical history, and physical examination will guide the clinician toward the appropriate etiology in many instances. One entity that may present with the complaint of low back pain is the cauda equina syndrome (CES), which is a relatively rare neurosurgical emergency.

Cauda equina syndrome is a serious neurologic disorder that is caused by compression of the conus medullaris or the spinal nerve roots comprising the cauda equina (Fig. 1). It is commonly associated with a triad of signs and symptoms including insensate buttocks and/or perineal areas (the so-called saddle anesthesia), loss of bowel or bladder function, and lower extremity weakness. Rapid recognition coupled with neurosurgical care will provide the patient with the best chance of recovery. We present a case of CES seen in the ED as an example of an uncommon but very severe etiology of low back pain (Table 1).

2. Illustrative case

A 39-year-old woman presented to the ED complaining of several hours of severe crampy abdominal pain. She stated that she had not been able to urinate or defecate for approximately 12 hours. In addition, she reported a 4-day history of low back pain since falling off of her motorcycle. She stated that her back pain had significantly resolved within the last day; however, she developed concomitant...
decreased sensation in both feet and difficulty walking. Her past medical history was significant for chronic low back pain. She was otherwise healthy.

Her physical examination was notable for an abdomen that was soft, diffusely tender to palpation, and with an obviously distended bladder. There were no peritoneal signs. Her neurologic examination was notable for 2+ bilateral patella deep tendon reflexes and absent bilateral ankle jerks. She had decreased sensation to pin-prick along her bilateral posterior buttocks and thighs. There was significant bilateral great toe, ankle, and quadriceps weakness. The straight leg raise (SLR) was positive bilaterally. She had a cautious, wide-based gait. The rectal examination revealed diminished anal sphincter tone with stool in the rectal vault. Her spine was nontender to palpation with no obvious step-offs, ecchymosis, or swelling.

The abdominal pain was presumed to be due to urinary retention, which was likely secondary to a cauda equine syndrome. A postvoid residual revealed a volume of 900 mL of urine. The patient was treated with intravenous (IV) steroids, a prompt neurosurgical consultation was obtained, and the patient underwent an urgent magnetic resonance imaging (MRI) (Figs. 2A, B), which revealed a large midline herniated disk at the L4 through L5 level. She was taken to surgery shortly thereafter. At surgery she was found to have a large central herniated disk at the L4 through L5 level with bilateral extremely tight and tense nerve roots and thecal sac. In addition, 2 large free disk fragments were found and removed from the disk space. At the time of discharge the patient had normal bowel function but required a Foley catheter to urinate. At 1 month follow-up the patient was evaluated in the urological clinic and found to have near normal voiding except for episodic bladder spasms which was confirmed with a normal voiding trial.

3. Discussion

3.1. Epidemiology/etiology

Cauda equina syndrome is rare with a prevalence among patients with low back pain of approximately 4 in 10000 [2].
Cauda equina syndrome usually arises from a massive midline disk herniation; however, it can also result from spinal metastases, spinal hematoma, epidural abscess, traumatic compression, or acute transverse myelitis [3]. Patel et al [4] reported CES resulting from abdominal aortic dissection. Cauda equina syndrome appears to be more prevalent in men within the fourth or fifth decade of life as they are most prone to disk herniation [5].

### 3.2. Pathophysiology

The most commonly affected disk space is L4-5 followed by L5-S1 and L3-4 [5]. The lumbar spine is anatomically composed of 5 lumbar vertebrae that are separated by the associated intervertebral disks. The cauda equina travels within the vertebral canal, which is bordered anteriorly by the posterior longitudinal ligaments that function to stabilize the vertebral bodies and disks. The localization of herniation to the lower lumbar disk makes anatomic sense because of posterior thinning of the longitudinal ligaments and the annulus fibrosis disk fibers which leads to weakened disk spaces [6]. It then follows that the weakened disk spaces help to promote herniation. This mechanism is illustrated in Figs. 3A-F.

### 3.3. Clinical presentation

The majority of patients (approximately 70%) have a history of chronic back pain as opposed to the other 30% of patients who present with CES as a primary manifestation of their herniated disk [5]. These patients usually present complaining of resolved or mild back pain secondary to some inciting event. Other complaints include bilateral sciatica, gait disturbances, frequent falling, insensate buttocks and/or feet, or even paralysis. On examination, combined motor and sensory deficits are hallmark physical findings and usually include bilateral leg weakness, positive SLR, decreased deep tendon reflexes, saddle anesthesia, and bowel/bladder retention or incontinence [2,3,5,6].

Diagnosis is usually made from the history and physical examination. If the diagnosis of CES is entertained, then 3 simple diagnostic maneuvers should be performed at the bedside. First, a rectal examination will assess perineal sensation and anal sphincter tone. Next, a postvoid residual should be obtained. If there is greater than 100 to 200 mL of residual urine, then urinary retention is likely present [6,7]. Finally, a SLR should be attempted to further evaluate for suspected radicular symptoms [6]. Urinary retention has a sensitivity of 90% and a specificity of about 95% [2] for the diagnosis of CES in those patients with the appropriate history and physical examination. If urinary incontinence is present, it is secondary to overflow incontinence from underlying acute urinary retention [5]. In addition, anal sphincter tone is diminished in up to 80% of patients [2].

### 3.4. Radiographic findings

The expedited radiographic examination is an important portion of the evaluation. Patients should undergo plain film...
radiography initially as fractures or other bony pathology may bring attention to areas of interest that will be delineated during further advanced radiographic studies [8]. Ideally, all patients with suspected CES should undergo urgent MRI of the spine for confirmation and localization of the lesion as it is the “gold standard” study. However, if MRI is not available, then computed tomography–myelography can be used as a more invasive alternative. The MRI should include the entire spine, as it is possible to have higher spinal lesions causing similar symptoms that would be missed on a focused MRI [3]. Furthermore, 10% of patients with spinal metastases have additional silent lesions located higher in the spine [3]. Diagnosis of these lesions is important for treatment planning [3].

3.5. Treatment

In the ED, treatment should include high-dose IV steroids and urgent surgical consultation. Empiric steroid treatment should be initiated as it is recommended in treatment of spinal compression secondary to tumor [3]. It may also help to relieve edema caused by the acute radiculopathy [6]. Dosing is controversial with recommendations ranging from 4 to 100 mg IV of dexamethasone [3].

Cauda equina syndrome has been historically managed as a surgical emergency; however, there is much controversy within the surgical literature regarding the urgency of operative intervention and the temporal relation of outcome [9]. Most authors agree that early surgical intervention is the best approach, and they recommend emergent surgery within 48 hours of symptom onset [5,10,11]. Patients who have earlier operations have decreased neurologic disability. Unfortunately, many patients are left with permanent deficits.

4. Summary

Cauda equina syndrome, although very rare, is a major source of severe morbidity from lower back pain. Prompt recognition of the associated historical features and neurologic dysfunction will lead to the diagnosis and treatment. The bedside diagnostic tests should include a rectal examination, postvoid residual bladder catheterization, and SLR. The radiographic study of choice remains MRI. Early steroid administration and surgical consultation are the desired ED treatment modalities. The major pitfall in diagnosis is not including CES in the back pain differential. To avoid missing the diagnosis, we suggest including screening questions for potential red flags in the history. These questions should include inquiries about the presence of sciatica, lower extremity paresthesias, bowel or bladder incontinence, saddle anesthesia, and fever. The major pitfall
in treatment is a delay in surgery. Therefore, prompt surgical consultation is advised so that operative intervention can be made in a timely fashion.

References

ED presentation of acute porphyria

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Received 27 March 2004; accepted 29 March 2004

Abstract Abdominal pain is a common complaint for visits to ED. Among the causes of abdominal pain, the acute porphyria may confuse emergency physicians. With wide range of unspecific symptoms and signs, acute porphyria is rarely considered as a differential diagnosis of acute abdomen in ED. Some patients even receive unnecessary surgery. There are 32 patients who visited the ED of National Taiwan University Hospital because of acute porphyric attacks over the past 13 years. Ten patients (3 males and 7 females) were diagnosed with acute porphyria for the first time at ED. The onset of age ranged from 17 to 55 years (mean, 32 years). All of our patients presented with abdominal pain but without fever, dermatologic, and neurologic symptoms that are typically presented in acute porphyria. On the average, most of them repeatedly sought for medical help because of persistent symptoms for 4 times before being definitely diagnosed and thus receiving the optimal treatment. Meanwhile, all patients needed at least 2 kinds of analgesic, and most of them needed narcotic analgesia for pain control before diagnosis. The most commonest point of tenderness is over epigastrium (7 of 10 patients). The laboratory and image studies of our patients were of no diagnostic value for acute porphyria, except for Watson-Schwartz test. In summary, our study revealed that when a patient after puberty with repetitive visits because of severe abdominal pain without reasonable causes and needs narcotics for pain control, acute porphyria should be taken into consideration.

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1. Introduction

Abdominal pain is a common cause for seeking help at ED. Once in a while, we may encounter such patients with repetitive episodes of severe abdominal pain. Clinically, the pain may be so severe that narcotic analgesics are needed to relieve the symptoms. At this time, it is necessary to seek the less common causes of severely unexplained abdominal pain, such as acute porphyria, before asking a psychological evaluation. Patients presenting with their first attack of porphyria may be misdiagnosed as other causes of acute abdomen. Thus, unnecessary anesthesia and laparotomy may result in a fatal outcome [1]. There are many medical literatures that have discussed the clinical manifestations of acute porphyria, but none of them focused on the acute
porphyric attack in the ED. In the present study, we retrospectively reviewed 13-year medical records to evaluate the clinical presentations of acute porphyria in ED. We analyzed their clinical manifestations and sought for any helpful clues for emergency physicians to make an early diagnosis of acute porphyria.

2. Methods

Clinical records of patients diagnosed with acute porphyrias at the National Taiwan University Hospital (NTUH) from 1990 to 2003 were retrospectively reviewed. A total of 32 patients were diagnosed with acute porphyria over the past 13 years. Patients who were initially diagnosed with acute porphyria at other hospitals, previous liver disease, and heavy metal poisoning were excluded from this study. Age, sex, laboratory study, image studies, times of visits, types of analgesic used, and treatment methods at ED were recorded. Gynecologic consultation has been done for all the female patients with lower abdominal pain. A fresh urine sample protected from light has been sent for Watson-Schwartz test [2]. The diagnosis of acute porphyria was made when the Watson-Schwartz test was positive along with the pertinent gastrointestinal or neurologic symptoms or signs and ruling out other specific diseases after laboratory and image studies. The results of laboratory studies were contemporary while the diagnosis of acute porphyria was made.

3. Results

A total of 32 medical records were reviewed. Ten patients (3 males and 7 females) were first diagnosed with acute porphyria at our ED over the past 13 years. The onset of age ranged from 17 to 55 years (mean, 32 years). All patients visited the ED because of abdominal pain. The most common tender area is over epigastrium (7 of 10 patients). All of our patients had neither fever nor skin lesions. However, none of them have neurologic symptoms on their ED visits. Two patients had seizures after admission. Eight patients without a family history of porphyrias repeatedly sought for medical help because of persistent symptoms (ranged from 2 to 8 times during an acute episode) before being accurately diagnosed and receiving the optimal treatment. Only 2 patients have a family history of acute intermittent porphyria and both of them were diagnosed during their first attack. Meanwhile, all patients needed at least 2 kinds of analgesias (mean, 2.6), and most of them needed narcotic analgesia for pain control before diagnosis (Table 1).

### Table 1 Clinical manifestations of acute porphyria on arrival at ED

<table>
<thead>
<tr>
<th>Case</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<th>8</th>
<th>9</th>
<th>10</th>
<th>Mean</th>
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<tbody>
<tr>
<td>Onset age (y)</td>
<td>29</td>
<td>55</td>
<td>27</td>
<td>43</td>
<td>17</td>
<td>24</td>
<td>42</td>
<td>22</td>
<td>34</td>
<td>30</td>
<td>32.3</td>
</tr>
<tr>
<td>Sex</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
<td>F</td>
<td>F</td>
<td>F</td>
<td>F</td>
<td>F</td>
<td></td>
</tr>
<tr>
<td>Location of abdominal pain</td>
<td>Epi</td>
<td>Luq</td>
<td>Epi</td>
<td>Epi</td>
<td>Epi</td>
<td>Low</td>
<td>Epi</td>
<td>Low</td>
<td>Epi</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<tr>
<td>Nausea</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Abdominal distension</td>
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<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<tr>
<td>Constipation</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Convulsion</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<td>Muscle weakness</td>
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<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Skin lesion</td>
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<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
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<tr>
<td>Fever</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
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</tr>
<tr>
<td>Total visits to ED, other hospitals, and OPD before diagnosis</td>
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<td>5</td>
<td>1</td>
<td>7</td>
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<td>6</td>
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<td>2</td>
<td>3</td>
<td>3</td>
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<td>4</td>
<td>2</td>
<td>2</td>
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<td>2.6</td>
</tr>
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<td>Use of narcotic analgesias</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td></td>
</tr>
<tr>
<td>Intravenous glucose</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>Heme arginate treatment</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td></td>
</tr>
</tbody>
</table>

Epi indicates epigastric area; Luq, left upper quadrant; and Low, lower abdomen.

### Table 2 The contemporary laboratory data of porphyrnic patients on arrival at ED

<table>
<thead>
<tr>
<th>Case</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (K/µL)</td>
<td>5.7</td>
<td>13.5</td>
<td>5.97</td>
<td>6.13</td>
<td>3.18</td>
<td>4.24</td>
<td>7.25</td>
<td>6.21</td>
<td>4.78</td>
<td>6.89</td>
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<tr>
<td>Segment (%)</td>
<td>74</td>
<td>81</td>
<td>62.4</td>
<td>71.4</td>
<td>38.4</td>
<td>89.1</td>
<td>54.6</td>
<td>64</td>
<td>75</td>
<td>76.5</td>
</tr>
<tr>
<td>Serum Na (mEq/L)</td>
<td>129</td>
<td>136</td>
<td>138.7</td>
<td>145</td>
<td>135</td>
<td>134.1</td>
<td>140.8</td>
<td>136</td>
<td>138</td>
<td>129.6</td>
</tr>
<tr>
<td>Serum K (mEq/L)</td>
<td>5.5</td>
<td>4.7</td>
<td>3.8</td>
<td>3.7</td>
<td>2.99</td>
<td>3.96</td>
<td>3.6</td>
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<td>3.87Y</td>
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<td>Urine color</td>
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<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>D</td>
<td>Y</td>
</tr>
</tbody>
</table>

WBC indicates white blood count; Y, yellowish; and D, dark.
The laboratory data of our patients were unremarkable for a specific diagnosis, including total white cell counts and differential counts, C-reactive protein, amylase, lipase, urine color, and liver function tests. Only 3 patients had hyponatremia (serum sodium level less than 135 mEq/L) when presenting to the ED. Only one patient presented with the characteristic dark urine (Table 2).

Plain abdomen, abdominal echo, upper gastrointestinal series, panendoscopy, and even abdominal computed tomography were performed when indicated. The image studies failed to demonstrate a unique picture of acute porphyric patients. All of our patients received intravenous 10% glucose for maintaining a high-energy intake after diagnosis of acute porphyria and they all responded to intravenous glucose therapy. Two patients (cases 5 and 6) received heme arginate after admission.

4. Discussion

Porphyria is referred to as group of diseases of the nervous system or skin (or both) that are associated with the generation of excess of porphyrin intermediates or their precursors because of decreased enzymatic activity during heme synthesis [1]. There are 4 types of porphyria in which acute attacks can occur: acute intermittent porphyria, variegate porphyria, hereditary coproporphyria, and δ-aminolevulinic acid dehydratase deficiency porphyria. The most frequently encountered are the first 2 types [3]. All 4 types are characterized by recurrent acute attacks, and their clinical manifestations include various presentation of severe abdominal pain, vomiting, hyponatremia, constipation, tachycardia, hypertension, muscle pain and weakness, seizures, paresis of the upper and lower extremities, paralysis and, probably, a variety of other neurologic and psychiatric symptoms [4]. Because the clinical course can vary from acute, self-limiting attacks to attacks that result in chronic or progressive deficits, the attacks may mimic many other psychological or medical disorders that may result in misdiagnosis [5]. It is very important to diagnose the disease rapidly. The precise type of acute porphyria is of secondary importance because the basic principles of management of the acute attack are the same, regardless of the specific diagnosis [6]. Thus, a positive screening test is the upmost method of screening and confirming the disease.

With the exception of the exceedingly rare δ-aminolevulinic acid dehydratase deficiency porphyria, the hallmark of the acute porphyric attack is increased excretion of the monopyrrole porphobilinogen in the urine. The “gold” standard for the estimation of urinary porphobilinogen is the method of Mauzerall and Granick [7]. This method is reliable and available commercially, but is slow and labor intensive and therefore unattractive to most clinical chemistry laboratories. Instead, most laboratories rely on simple qualitative “screening tests” (eg, the Watson-Schwartz test) in which urine is reacted directly with Ehrlich’s reagent and this method is used at our hospital.

According to symptoms of acute porphyria in the present study, including abdominal pain, vomiting, nausea, abdominal distension, constipation, convulsion, and muscle weakness, acute attacks are more common in women than in men. Attacks are most frequent during the second to fourth decades and rarely before puberty. Both results are compatible with other studies [8]. But neither sensory loss, psychological symptoms (such as abnormal behaviors, confusion, agitation, or hallucinations) nor skin lesions were found in our patients. Only 2 patients had neurologic presentations in our study. But the neurologic presentations both occurred after admission. Besides, in the present study, the incessant pleas of affected patients for narcotic analgesias might lead to unwarranted suspicion of addiction. However, as the attack remitted, the need for narcotic analgesias disappeared. The pain of acute porphyria is notorious for simulating appendicitis, intestinal obstruction, and renal or gallbladder colic. This, coupled with the slight leukocytosis and low-grade fever that sometimes occur, has caused some patients to undergo unnecessary laparotomy [9].

After comparing the results of the image and laboratory studies in the present study, except the Watson-Schwartz test, we failed to demonstrate other helpful modality to diagnose acute porphyria. Otherwise, we found one interesting laboratory finding—initial presentation of hyponatremia is only found in 3 patients, but if we followed up the serum sodium level within 48 hours, the case number of patients with hyponatremia would be up to 6. Hyponatremia may be due to vomiting, inappropriate fluid therapy, or the syndrome of inappropriate antidiuretic hormone release [1]. Whether the dynamic serum sodium change is of any diagnostic value, further investigations should be done to get the conclusions.

The treatment of all acute porphyrias is essentially identical. Treatment between attacks comprises adequate nutritional intake, avoidance of drugs and chemicals known to exacerbate porphyria, and prompt treatment of other intercurrent diseases or infections [10]. During acute attacks of porphyria, oral and intravenous glucose (for maintaining a high-energy intake) and heme arginate are the mainstay of treatment [11]. They reduce synthesis of aminolevulinic acid, resulting in a clinical and biochemical remission, with urinary excretion of aminolevulinic acid and porphobilinogen falling toward normal values.

When evaluating patients with repetitive episodes of severe abdominal pain without a reasonable cause, we advocate maintaining a high degree of awareness for porphyria. Without carefully gathering a history and having a high index of clinical suspicion, it will be delayed to make the correct diagnosis. In turn, it possibly results in increasing morbidity and consuming the medical and financial resources.

In summary, in our study, it was revealed that when a patient after puberty repeatedly visits ED because of severe
abdominal pain without reasonable causes and needs narcotics for pain control, acute porphyria should be taken into consideration. The Watson-Schwartz test may be a quick screening test. Besides, acute porphyria in ED may be without neurologic or dermatologic presentations, which may be encountered more in the outpatient department. During acute attacks of porphyria, oral and intravenous glucose (for maintaining a high-energy intake) and heme arginate are the mainstay of treatment.

References


Late postpartum eclampsia as an obstetric complication seen in the ED

Brendon Graeber B.A., Tamara Vanderwal B.Sc., Robert J. Stiller M.D.,*, Michael J. Werdmann M.D.

Abstract Preeclampsia is a complication of pregnancy associated with hypertension and proteinuria. Preeclampsia may be associated with grand mal seizures and is termed eclampsia. Historically, eclampsia occurring more than 48 hours after delivery, known as late postpartum eclampsia, was thought to be uncommon; however, recent evidence suggests that its incidence is increasing. In addition, the presentation of late postpartum preeclampsia-eclampsia may differ from that occurring during the pregnancy. This contributes to difficulty in diagnosing late postpartum preeclampsia-eclampsia in an emergency department setting. We report 2 cases of late postpartum eclampsia presenting 8 days after delivery, which highlight the unique features of this disorder and discuss some of the difficulties in managing these patients. Greater awareness and knowledge of this disorder by ED physicians should improve outcomes in these potentially life-threatening cases.

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1. Introduction

Hypertensive disorders of pregnancy occur in 5% to 10% pregnancies [1]. Preeclampsia is defined as blood pressure higher than 140/90 mm Hg and proteinuria of more than 300 mg per 24 hours occurring after 20 weeks of gestation [2]. Edema may also accompany this disorder along with abnormalities in coagulation or liver function testing. In its most severe form, preeclampsia may be accompanied by new-onset grand mal seizures and is termed eclampsia. Preeclampsia-eclampsia generally occurs close to term and typically resolves within 48 hours of delivery. Because this disorder is most commonly seen in either pregnant or immediately delivered women, obstetricians are most familiar with its presentation and treatment. However, preeclampsia-eclampsia may develop after 48 hours and patients may initially present to the emergency department (ED) with the findings of hypertension and headaches, with later development of seizures. We present 2 cases of late postpartum eclampsia seen initially in the ED and review the pertinent features of this disorder.

2. Case 1

A 27-year-old woman, gravida 2, para 1, presented to the ED with complaints of headache and visual changes with a
duration of 1 day. Eight days earlier, she had delivered a full-term baby boy by cesarean section, which was indicated because of failure to progress in labor after an uncomplicated pregnancy. Her headache was localized to the left side, and the patient reported the pain at 7/10. Her initial blood pressure was 177/92 mm Hg, which decreased to 140/68 mm Hg over the next few hours in the ED. On evaluation by the ED physician, the pain was not relieved by acetaminophen or ibuprofen. She reported a history of migraines in the distant past. Her neurological examination was reported as normal. A cranial computerized tomogram without contrast was unremarkable. The urine analysis for protein was negative. The patient was given 2 doses of meperidine and the pain resolved. She was discharged approximately 4 hours later.

At home, she experienced vomiting and the return of her headache. Approximately 90 minutes after discharge, her mother reported a loss of consciousness, followed by convulsions lasting about 5 minutes, with postictal confusion. She experienced a second seizure at home approximately 5 minutes later. She arrived in the ED by ambulance at approximately 2 hours after her initial discharge. Diazepam and labetolol were administered, a lumbar puncture was performed, and neurology and obstetric consultations were requested. Laboratory results included the following values: creatinine, 0.8 mg/dL; aspartate aminotransferase, 30 IU/L; hemoglobin, 11.1 mg/dL; hematocrit, 33.9; and platelets, 427,000. The urine protein/creatinine ratio was 0.18 (normal <0.3).

The patient was evaluated by the obstetrics consultant who recommended that magnesium sulfate therapy be instituted, with an initial 6-g intravenous bolus. She experienced a third generalized convulsion lasting about 60 seconds shortly after beginning the intravenous magnesium sulfate infusion. After this episode, the patient was continued on magnesium sulfate and experienced no further seizures.

The patient was admitted to the medical intensive care unit. Blood cultures, electroencephalogram, and magnetic resonance imaging studies were ordered and the results were normal. Cerebrospinal fluid cultures were also negative. The patient had no further complications and was discharged on hospital day 2.

### 3. Case 2

A 26-year-old woman, gravida 1, para 1, who had delivered a healthy full-term baby by spontaneous vaginal delivery, presented to the ED in the evening of the eighth postpartum day complaining of worsening, persistent headache with a duration of 2 days, accompanied by nausea and vomiting with some blurring of her vision. Initially, the headache had responded to acetaminophen, but on the day of admission, nothing had succeeded in relieving it. Examination of the patient revealed only a significantly elevated blood pressure of 166/94 mm Hg. The patient denied other complaints or pain beyond her headache. Given her postpartum status, an obstetrics consultation was requested.

The obstetrics consultant suspected postpartum pre-eclampsia, and magnesium sulfate was requested from the pharmacy. Approximately 3 hours later, while awaiting the institution of magnesium sulfate therapy, the patient experienced a generalized tonic-clonic seizure, accompanied by urinary incontinence. She received diazepam (10 mg IV). After the resolution of her first seizure, a blood chemistry panel and a cranial computerized tomography (CT) study were ordered by the ED physician. Half hour after her first seizure, the patient experienced another tonic-clonic seizure in the ED. Magnesium sulfate therapy was then initiated with a 6-g intravenous bolus followed by a 2-g/h intravenous infusion, and she underwent a CT scan. CT findings were negative for any obvious abnormalities. The patient was admitted to the medical intensive care unit receiving intravenous magnesium sulfate with a diagnosis of late postpartum eclampsia.

The patient’s blood chemistry results were normal including the following values: Na+, 147 mEq/L; Cl−, 109 mEq/L; HCO3−, 14 mEq/L; K+, 4.0 mEq/L; blood, urea, and nitrogen, 11 mg/dL; creatinine, 0.7 mg/dL; Ca2+, 9.5 mg/dL; and glucose, 70 mg/dL. However, a complete blood count revealed a hemoglobin level of 14.0 g/dL, a hematocrit level of 43.2, a platelet count of 89,000, and a white blood cell count of 19,100. Spot urinalysis showed 1+ protein.

The patient’s remaining hospital course was unremarkable. Cranial magnetic resonance imaging was negative. Blood cultures were negative as well. A urine culture was positive for Klebsiella pneumoniae, which was treated with antibiotic therapy. Intravenous magnesium sulfate infusion at 2 g/h was continued for 48 hours. The remainder of her hospital admission was uncomplicated and she was discharged on the third day.

### 4. Discussion

Preeclampsia is a complication of pregnancy consisting of hypertension and proteinuria. In its most severe form, preeclampsia may be accompanied by seizures and is termed eclampsia. Generally, eclampsia occurs either before or within 48 hours of delivery. Eclampsia occurring more than 48 hours but less than 4 weeks after delivery is known as late postpartum eclampsia. Late postpartum eclampsia was initially thought to be very uncommon [3]. However, recent data suggest that the timing of eclampsia in this country is changing and that the incidence of documented late postpartum eclampsia is increasing [3,4].

Lubarsky et al [3] reported on 334 cases of eclampsia from 1977 to 1992. Ninety-seven cases (29%) occurred in postpartum period. Of 97 cases of postpartum eclampsia, 54 (56%) occurred after 48 hours of delivery. Convulsions occurred 3 to 23 days postpartum, with mean of 6 days postpartum. Of 54 patients, 37 (69%) developed convulsions.
after hospital discharge and 45 (83%) complained of either severe headache (n = 38) or visual disturbances (n = 17) before seizures occurred. In 56% of cases, preeclampsia was diagnosed before the onset of convulsions.

Chames et al [4] studied patients with eclampsia at 3 centers during the period of 1996-2001. There were 89 patients diagnosed with eclampsia. Twenty-nine (33%) cases occurred in the postpartum period. Of 29 patients with postpartum eclampsia, 23 (79%) occurred after 48 hours. The presenting symptoms were headache (87%), visual changes (44%), nausea or vomiting (22%), and epigastric pain (22%). Only 5 patients of these 23 patients (22%) were previously diagnosed with preeclampsia during labor.

The differential diagnosis of seizures in a postpartum patient includes cerebral venous thrombosis, intracerebral hemorrhage, hypertensive encephalopathy, space-occupying lesions of the brain, metabolic disorders such as hypoglycemia, hyponatremia, and epilepsy. Imaging studies of the brain are recommended when focal or persistent neurological findings are present.

The recommended treatment of preeclampsia for prevention of seizures is magnesium sulfate [1,2]. This is administered as a 4- to 6-g intravenous infusion over 15 to 30 minutes, followed by a maintenance dose of 2 g/h [1]. The maintenance infusion should be titrated to the patient’s patellar reflexes, urine output, respiratory rate, and serum magnesium levels [1,2]. Magnesium toxicity should, thus, be avoidable, but can be treated with parenteral calcium gluconate (10 mL of a 10% solution), if necessary. Blood pressure higher than 160/110 mm Hg is generally treated with antihypertensive agents such as labetolol, hydralazine, or nifedipine. Treatment with magnesium sulfate is generally discontinued after 24 hours.

In contrast with the nonpregnant patient, magnesium sulfate is the preferred agent for prevention and treatment of seizures due to eclampsia. Lucas et al [5] compared magnesium sulfate and phenytoin for the prevention of eclamptic seizures. In this study, 10 of the 1089 women randomized to phenytoin had eclamptic seizures whereas none of the 1049 women randomized to magnesium sulfate experienced convulsions. This provided clear evidence of the superiority of magnesium sulfate over phenytoin for this disorder.

Late postpartum preeclampsia may not present with all the classical symptoms of intrapartum preeclampsia such as hypertension (≥140/90), proteinuria, and associated symptoms such as headache, visual changes, abdominal pain, or edema. Laboratory abnormalities, such as hemolysis, elevated liver transaminases, and low platelet count, known as the HELLP syndrome, are seen in a minority of cases of late postpartum eclampsia [4]. This makes the diagnosis of late postpartum preeclampsia-eclampsia more difficult to make.

Our 2 cases demonstrate the subtlety with which late postpartum preeclampsia may present. In both cases, the presentation was marked by hypertension associated with worsening headache and visual changes with nausea and vomiting. However, there were no signs of preeclampsia during labor or during the immediate postpartum period and symptoms did not develop until after discharge. Significant proteinuria, a hallmark of preeclampsia, was not present in the first case. The second case did show laboratory abnormalities including hemoconcentration, thrombocytopenia, and proteinuria.

In conclusion, patients may present after hospital discharge and be seen by either primary care and emergency physicians, and it is important for other specialties to recognize women at risk for late postpartum preeclampsia-eclampsia. Prompt treatment of preeclampsia may prevent the progression to seizures. Magnesium sulfate, the preferred therapy, may not be stocked in the ED, leading to a potential delay in instituting therapy once ordered. Obstetricians should inform new mothers about the symptoms of postpartum preeclampsia, such as the development of severe headache, visual changes, or abdominal pain. In addition, emergency physicians should always be prepared to consider late postpartum preeclampsia in postpartum hypertensive patient.

Acknowledgment

The authors thank Steven A. Laifer, MD, for his careful reading and review of the manuscript.

References

The electrocardiographic toxidrome: the ECG presentation of hydrofluoric acid ingestion

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Received 23 April 2004; accepted 24 April 2004

Abstract The clinician can approach the poisoned patient using the toxidrome system of toxin identification; this approach makes use of findings noted on the physical examination, highlighting the importance of abnormalities in blood pressure, heart rate, respiratory effort, body temperature, mental status, pupillary size, skin color, diaphoresis, and gastrointestinal sounds. Such a method provides structure and guidance to the clinical evaluation, providing the clinician with rapid diagnostic information and suggesting urgent management issues. A case of hydrofluoric acid poisoning is used as an example of this diagnostic approach. The patient demonstrated systemic toxicity accompanied by oral irritation and electrocardiographic abnormality (QRS complex widening and QT interval prolongation). The constellation of these findings suggested the possibility of a caustic agent (history and examination) with potential effect on potassium and calcium metabolism (electrocardiographic abnormalities). Such a constellation strongly suggested hydrofluoric acid as the culprit toxin.

1. Introduction

The clinician can approach the poisoned patient using the toxidrome system of toxin identification. Such a method provides structure and guidance to the clinical evaluation, providing the clinician with rapid diagnostic information and suggesting urgent management issues. The toxidrome method emphasizes the use of the physical examination, highlighting the importance of abnormalities in blood pressure, heart rate, respiratory effort, body temperature, mental status, pupillary size, skin color, sweat presence, and gastrointestinal sounds.

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We present a case of hydrofluoric acid poisoning which initially presented as an unknown ingestion. The patient demonstrated systemic toxicity accompanied by oral irritation and electrocardiographic abnormality (QRS complex widening and QT interval prolongation). The constellation of these findings suggested the possibility of a caustic agent (history and examination) with potential effect on potassium and calcium metabolism (electrocardiographic abnormalities). Such a constellation strongly suggested hydrofluoric acid as the culprit toxin. Such rapid recognition of these findings enabled the clinician to provide specific life-saving therapy in rapid fashion to the patient. With this case, we review the electrocardiographic differential diagnosis of QRS complex widening and QT interval prolongation, suggesting that such findings be incorporated into the clinician’s toxidrome.
evaluation strategy in the initial management of the poisoned patient.

2. Case presentation

A previously healthy 47-year-old man accidentally ingested a blue liquid he thought was a sport drink. He immediately noted throat irritation. Within five minutes of the ingestion, he developed nausea and vomiting. He presented to the emergency department (ED) within 1 hour of the ingestion with a complaint of nausea, weakness, and intense pleuritic chest pain. His initial vital signs revealed the following: temperature 34.5°C, pulse 130 beats/min, blood pressure 102/66 mm Hg, and respiratory rate 20 breaths/min. His voice was hoarse and he had difficulty swallowing his secretions. His oropharynx revealed erythema without edema or ulceration. Auscultation of his chest revealed diffuse rhonchi and tachycardia without murmurs, rubs, or gallops. His abdomen was soft with mild tenderness diffusely and audible hyperactive bowel sounds. His skin was warm and diaphoretic. He was alert, orientated, and demonstrated good strength throughout without tremor or clonus.

His initial electrocardiogram (ECG) 1 hour after ingestion revealed a sinus tachycardia with a QRS complex duration of 110 milliseconds and prominent T waves (Fig. 1). His initial arterial blood gas revealed the following: pH 7.28, PCO₂ 29, PO₂ of 209, and HCO₃ 13. Within one-half hour of his arrival, he became increasingly agitated and his systolic blood pressure dropped to 80. A repeat ECG (Fig. 2) showed an increased widening of the QRS complex to 152 milliseconds, a QTc interval of 742 milliseconds, and further peaking of the T waves. Initial bedside evaluation of the ingested fluid by litmus paper revealed a pH less than 4.0.
Based upon the apparent caustic nature of the ingestion with an acidic pH and electrocardiographic abnormalities suggestive of calcium and potassium effect, hydrofluoric acid ingestion was suspected. The patient received intravenously 4 g of calcium gluconate, 4 g of magnesium sulfate, 2 g of calcium chloride, and 200 mEq of sodium bicarbonate over the ensuing 30 minutes. His blood pressure increased to 158/94 with associated QRS complex narrowing to 102 milliseconds (Fig. 3). Initial laboratory values, drawn upon the patient’s arrival, were remarkable for a white blood cell count of 12.5 × 10^9/L, hematocrit of 49%, platelet count of 191 × 10^9/L; sodium was 138 mmol/L, potassium 5.7 mmol/L, chloride 103 mmol/L, bicarbonate 12 mmol/L, urea nitrogen 10 mg/L, creatinine 1.4 mg/L, glucose 193 mg/L, calcium less than 4.0 mmol/L, and magnesium 0.7 mmol/L.

His electrolyte abnormalities and metabolic acidosis resolved within 12 hours of admission after receiving an additional 3 g of calcium chloride, 6 g of magnesium sulfate, 200 mEq of sodium bicarbonate, and 15 mmol of sodium phosphate. The electrocardiographic abnormalities resolved within 12 hours (Fig. 4). He was intubated within the first 24 hours, however, secondary to progressive respiratory distress and hypoxemia. His chest radiograph revealed acute interstitial changes consistent with aspiration pneumonia. The patient ultimately was discharged after a 21-day hospitalization complicated by gastrointestinal bleeding, pneumonia, tracheal stenosis requiring placement of a tracheostomy, and pulmonary embolism requiring placement of an inferior vena cava filter.

Further laboratory evaluation of the ingested liquid revealed a pH of 0.8. The fluid was identified as hydrofluoric acid through flame ionization and atomic absorption.

3. Discussion

In this case, the patient ingested an unknown liquid substance. His initial symptoms of throat burning, emesis,
difficulty with phonation, inability to manage secretions, and chest and abdominal pain were consistent with a caustic ingestion. A rapid bedside litmus paper test revealed that the substance had an acidic pH. An initial arterial blood gas revealed a metabolic acidosis with concomitant respiratory alkalosis. The patient subsequently progressed to hypotension with electrocardiographic changes of both QRS complex widening and QT interval prolongation. For the astute clinician who considers the differential diagnosis for an acidic agent that produces metabolic acidosis, hypotension, and electrocardiographic abnormalities (QRS complex widening and QT interval prolongation), the diagnosis is readily determined to be hydrofluoric acid ingestion.

Hydrofluoric acid is utilized in many industrial settings for the production of integrated circuits, fluorides, plastics, germicides, insecticides, and for the etching and cleaning of silicone, glass, metal, stone, and porcelain. Hydrofluoric acid–containing products are sold as automotive cleaning products in local stores. In 2002, just over 1000 hydrofluoric acid exposures were reported to US poison centers, with 23 major outcomes and 5 deaths reported [1]. Hydrofluoric acid rapidly corrodes and penetrates skin and mucous membranes. Ingestion of hydrofluoric acid may result in local mucosal caustic effects, nausea, vomiting, abdominal pain, and hemorrhagic gastritis. Systemic electrolyte abnormalities may occur. The absorbed fluoride ions rapidly bind to available calcium and magnesium ions, decreasing the body’s levels of these divalent cations. Hyperkalemia often follows due to an efflux of potassium out of cells into the extracellular space.

There are numerous causes of QRS prolongation. The electrocardiographic differential diagnosis of wide complex tachycardia classically includes ventricular tachycardia vs paroxysmal supraventricular tachycardia with aberrant ventricular conduction. Aberrant ventricular conduction may be due to a preexisting bundle branch block (BBB), a functional (rate-related) bundle malfunction resulting in a widened QRS complex when the heart rate exceeds a characteristic maximum for that patient, or accessory atrioventricular conduction as encountered in preexcitation syndromes, such as that described by Wolff, Parkinson, and White. Other clinical syndromes less frequently encountered in this wide complex tachycardia differential, although very important to the acute care physician, include scenarios related to marked sinus tachycardia with preexisting BBB configuration, recent electrical cardioversion, and ischemic electrocardiographic forms (the giant R wave) misinterpreted as a widened QRS complex with tachycardia.

In a “toxicologic” setting, QRS complex widening likely results directly from sodium channel blockage or indirectly from toxin-induced hyperkalemia. Direct toxin-induced blockade of cardiac sodium channels will cause QRS complex widening, also known as a membrane stabilizing effect [2,3]. Cardiac voltage-gated sodium channels reside in the cell membrane and open in response to depolarization of the cell. The sodium channel blockers bind to the transmembrane sodium channels and decrease the number available for subsequent depolarization. This phenomenon creates a delay of sodium entry into the cardiac myocyte during phase 0 of the cardiac depolarization. As a result, the upstroke of depolarization is slowed and the QRS complex widens (Fig. 5).

Fig. 5 Action potential with accompanying ECG. Note the effects of sodium channel blockade with QRS complex widening and potassium efflux blockage with QT interval prolongation.

### Table 1: Cardiac sodium channel blocking agents

<table>
<thead>
<tr>
<th>Agent</th>
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<tbody>
<tr>
<td>Amantadine</td>
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<tr>
<td>Amitriptyline</td>
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<tr>
<td>Amoxapine</td>
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<tr>
<td>Carbamazepine</td>
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<tr>
<td>Chloroquine</td>
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<tr>
<td>Cocaine</td>
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<tr>
<td>Desipramine</td>
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<tr>
<td>Diltiazem</td>
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<tr>
<td>Diphenhydramine</td>
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<tr>
<td>Disopyramide</td>
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<tr>
<td>Doxepin</td>
</tr>
<tr>
<td>Encaidenide</td>
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<tr>
<td>Flecaïnide</td>
</tr>
<tr>
<td>Hydroxychloroquine</td>
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<tr>
<td>Imipramine</td>
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<tr>
<td>Loxapine</td>
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<tr>
<td>Maprotiline</td>
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<tr>
<td>Moricizine</td>
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<tr>
<td>Nortriptyline</td>
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<tr>
<td>Orphenadrine</td>
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<tr>
<td>Phenothiazines</td>
</tr>
<tr>
<td>Procainamide</td>
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<tr>
<td>Propranolol</td>
</tr>
<tr>
<td>Propafenone</td>
</tr>
<tr>
<td>Propoxyphene</td>
</tr>
<tr>
<td>Saxitoxin</td>
</tr>
<tr>
<td>Tetrodotoxin</td>
</tr>
<tr>
<td>Thiopental</td>
</tr>
<tr>
<td>Quinidine</td>
</tr>
<tr>
<td>Quinine</td>
</tr>
<tr>
<td>Verapamil</td>
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</tbody>
</table>
Sodium channel blockers not only result in widening of the QRS complex—other abnormal QRS complex configurations are also possible. In some cases, the QRS complexes may assume the pattern of recognized BBBs. In the most severe cases, the QRS complex widening becomes so profound that the ultimate origin of the rhythm disturbance is impossible; in fact, it is difficult to distinguish between ventricular and supraventricular rhythms in this clinical scenario [4]. Continued widening of the QRS complex may result in a sine wave pattern and, eventually, asystole. Sodium channel blockers may also induce a monomorphic ventricular tachycardia. It has been theorized that the sodium channel blockers can cause slowed intraventricular conduction, unidirectional block, and the development of a reentrant circuit, ultimately resulting in ventricular tachycardia. This dysrhythmia can then degenerate into ventricular fibrillation.

The agents listed in Table 1 are similar in that they may induce myocardial sodium channel blockade.

Myocardial sodium channel blocking drugs comprise a diverse group of pharmaceutical agents. As a result, patients poisoned with these agents will have a variety of clinical presentations. For example, the following agents—tricyclic antidepressants, propoxyphene, and cocaine—are all potent sodium channel blockers; yet these same agents will produce other findings (anticholinergic, opioid, and sympathomimetic syndromes, respectively) as well.

The other major mechanism of QRS complex widening in this case is hyperkalemia. The earliest electrocardiographic sign of hyperkalemia is the appearance of tall, symmetric T waves (Fig. 6); this T-wave morphology is described as “hyperacute” and may be confused with the hyperacute T wave of early transmural myocardial infarction. As the serum potassium level increases, the T waves tend to become taller, peaked, and narrowed in a symmetric fashion in the anterior distribution. With further increases in the serum concentration, the PR interval is prolonged followed eventually—and most ominously—with QRS complex widening (Fig. 6). Ultimately in hyperkalemia, the QRS complex assumes a sine wave configuration and the rhythm is described as sinoventricular. At any point or time in this pathophysiologic worsening, ventricular fibrillation may appear.

This patient also manifested progressive QT interval prolongation which may result from either toxin-induced electrolyte alterations or direct myocardial effects of the poison. Electrolyte abnormalities associated with QT interval prolongation include hypokalemia, hypomagnesemia, and hypocalcemia. QT interval prolongation may also be due to toxin-induced blockade of potassium efflux channels during phase 3 of the action potential corresponding with repolarization (Fig. 5). These events may place the patient at risk for polymorphic ventricular tachycardia or torsades de pointes [5]. Some medications, such as sotalol, are prescribed specifically for this mechanism [6]. Other medications possess this activity as an unwelcome side effect at therapeutic doses, such as cisapride and terfenadine, which have been removed from the North American market because of reports of sudden cardiac death [7,8]. Other medications have rarely been reported to cause QT interval prolongation except when taken in massive overdose. A number of chemicals found within the home and workplace are also associated with QT interval prolongation. A complete listing of the reported agents associated with QT interval prolongation is noted in Table 2 [9].

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Cardiac potassium efflux channel blocking agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Arsenic</td>
</tr>
<tr>
<td>Astemizole</td>
<td>Chloroquine</td>
</tr>
<tr>
<td>Cisapride</td>
<td>Cyclic antidepressants</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>Disopyramide</td>
</tr>
<tr>
<td>Droperidol</td>
<td>Erythromycin</td>
</tr>
<tr>
<td>Flecaïnide</td>
<td>Haloperidol</td>
</tr>
<tr>
<td>Hydroxychloroquine</td>
<td>Ketoconazole</td>
</tr>
<tr>
<td>Organophosphates</td>
<td>Pentamidine</td>
</tr>
<tr>
<td>Phenothiazines</td>
<td>Procainamide</td>
</tr>
<tr>
<td>Quinidine</td>
<td>Quinine</td>
</tr>
<tr>
<td>Quinine</td>
<td>Sotalol</td>
</tr>
<tr>
<td>Sotalol</td>
<td>Terfenadine</td>
</tr>
<tr>
<td>Terfenadine</td>
<td>Venlafaxine</td>
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</tbody>
</table>
All patients presenting with signs and symptoms consistent with hydrofluoric acid ingestion should be aggressively managed. The patient’s airway should be patent and adequate ventilation assured. If necessary, endotracheal tube intubation should be performed early before edema leads to airway obstruction. The patient should be placed on continuous cardiac monitoring with pulse oximetry and frequent neurological checks should be made. The initial treatment of hypotension consists of intravenous fluids, followed by pressors as needed. The patient’s pulmonary status should be monitored closely for clinical signs consistent with pulmonary aspiration. Activated charcoal, syrup of ipecac, and gastric lavage are absolutely contraindicated in patients who have ingested caustics. Serum electrolytes should be obtained hourly and include serial calcium, magnesium, and potassium levels. The clinician should obtain serial ECGs looking for signs of hypocalcemia (prolonged QTc interval) and hyperkalemia (peaked T waves). Large amounts of calcium and magnesium may be needed to normalize serum levels. fluoride-induced hyperkalemia has been reported to be difficult to reverse. Early aggressive therapy with glucose, insulin, and/or sodium bicarbonate may be effective. Quinidine has been shown to be effective in preventing the K+ efflux from cells and preventing cardiotoxicity in fluoride-toxic dogs [11].

4. Conclusion

This patient manifested findings consistent with caustic ingestion and concomitant electrocardiographic findings including sinus tachycardia, QRS complex prolongation, and QT interval prolongation. Only hydrofluoric acid ingestion would account for all the findings noted in this case. Because of its acidic nature, hydrofluoric acid can result in nausea, vomiting, abdominal pain, chest pain, inability to swallow secretions, gastrointestinal bleeding, voice changes, and respiratory distress. Hydrofluoric acid ingestion also could result in electrolyte abnormalities. The fluoride ions chelate serum calcium and magnesium, thereby leading to precipitous drops of both of these electrolytes [10]. The subsequent hypomagnesemia and hypocalcemia can lead to prolongation of the QT interval. Published reports also have noted the development of hyperkalemia after significant hydrofluoric exposures [11,12] This hydrofluoric acid–induced hyperkalemia can subsequently result in prolongation of the QRS complex. The treatment of the above toxicity consists of supportive care for the caustic effects and correction of electrolyte and fluid abnormalities.

References

Pneumatosis intestinalis and hepatic portal venous gas after CPR

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Received 10 May 2004; accepted 10 May 2004

Abstract Pneumatosis intestinalis and hepatic portal venous gas are usually associated with severe intra-abdominal pathologies. As diagnostic technologies advanced, a number of variant etiologies have been identified. We report 2 cases in which pneumatosis intestinalis and hepatic portal venous gas developed after prolonged cardiopulmonary resuscitation (CPR). The pathogenic mechanism was most probably bowel infarction caused by poor mesenteric perfusion during and after CPR. Limited cardiac output during prolonged resuscitation and severe vasoconstriction after large doses of epinephrine and vasopressors might both contribute to the compromised mesenteric perfusion. The risk seems especially high for old patients with severe atherosclerosis. Once it happens, the prognosis is extremely poor. In patients of cardiac arrests receiving prolonged CPR, catastrophic complications like this should be considered in the postresuscitation phase, especially those with multiple risk factors like old age, severe atherosclerosis, and use of large doses of vasoconstrictors.

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1. Introduction

Pneumatosis intestinalis (PI) and hepatic portal venous gas (HPVG) are usually associated with severe intra-abdominal diseases. Emergency explorative laparotomy is often needed because, if not treated properly, high mortality is anticipated \cite{1,2}. In recent years, the diagnostic rate of PI and HPVG increased with the advances in diagnostic technologies. A number of variant etiologies were also identified \cite{3,4}. We reported 2 cases in which PI and HPVG developed after prolonged resuscitation for cardiac arrest. To our knowledge, this etiology has not been reported in the English literature before.

2. Case reports

2.1. Case no. 1

An 81-year-old man with dilated cardiomyopathy was sent to the ED with the presentation of sudden cardiac arrest...
in the early morning. Cardiopulmonary resuscitation (CPR) was started on the scene and 6 times of defibrillation was done by automated external defibrillator because of refractory ventricular tachycardia. On arrival at the ED, CPR was continued because of relapsing pulseless ventricular tachycardia and asystole, and 22 more countershocks were delivered. Return of spontaneous circulation was achieved after 42 minutes of resuscitation at the ED. However, the patient’s consciousness was not restored. A total of 25 mg epinephrine, 40 U vasopressin, 200 mg lidocaine, and 150 mg amiodarone were administered during the resuscitation efforts. He was then admitted to the intensive care unit (ICU) for postresuscitation care.

At the ICU, sinus rhythm was maintained under amiodarone infusion (900 mg for 24 hours), and hemodynamics was stabilized with the use of dopamine (15 μg/kg per minute). Echocardiogram revealed global hypokinesia of the left ventricle with ejection fraction of 21%. The initial biochemical studies were not remarkable except elevation of serum creatinine (1.7 mg/dL). Abdominal echographic screening on the first day postresuscitation showed no specific findings. However, progressive abdominal distension and elevation of serum AST (391 U/L), ALT (93 U/L), and amylase (255 U/L) were noted in the next morning. Follow-up abdominal echogram showed remarkable gas in the portal veins (Fig. 1A). Abdominal computed tomography (CT) was then performed, which, in addition to HPVG (Fig. 1B), revealed marked dilatation of the bowel loops with air in the intestinal wall (Fig. 1C). Severe atherosclerosis of the abdominal aorta with involvement of the superior mesenteric artery orifice was also noted (Fig. 1D). Bowel infarction with PI and HPVG was diagnosed. Surgeon was consulted but surgical intervention was not done because of poor general condition. He received supportive treatment and died on the third day because of multiple organ failure.

Fig. 1  A, Abdominal echogram showed scattered echogenic patches within the liver parenchyma with hepatopetal flow of the echogenic patches (arrowheads) cascading up in the portal veins. B, Abdominal CT revealed gas in the peripheral branches of the portal vein (short arrows). The gas was distributed distally within 2 cm of the liver capsule, differentiating itself from pneumobilia. C, The abdominal aorta showed marked calcification with involvement of the superior mesenteric artery orifice. Gas was also noted within the superior mesenteric vein (long arrow). D, Marked PI presented as air within dependent and nondependent parts of the bowel wall.
2.2. Case no. 2

A 57-year-old man with liver cirrhosis and hepatoma presented to the ED because of hepatic encephalopathy. The initial physical examination was not remarkable except flapping tremors. Unfortunately, sudden-onset massive hematemesis followed by circulatory collapse was noted while he stayed in the observation room. Cardiopulmonary resuscitation was initiated promptly, and return of spontaneous circulation was achieved 24 minutes later after epinephrine and large amount of fluid resuscitation. He was then admitted to the ICU for continued fluid and blood component therapy. Unfortunately, CPR and defibrillation were repeated several times in the following hours because of recurrent ventricular fibrillation. Totally, 11 times of defibrillation (3420 J) and 15 mg of epinephrine were given during resuscitation. Intravenous dopamine, somatostatin, and amiodarone infusion were also maintained. Laboratory examinations after admission showed metabolic acidosis, hypokalemia, and hypocalcemia. As fever and abdominal distension developed later, abdominal echogram was done 12 hours postresuscitation, which revealed dilatation of the ascending colon with air entrapped in the bowel wall (Fig. 2A). Under the diagnosis of bowel infarction with PI, surgeon was consulted for possible surgical exploration. However, operation was considered impractical because of high surgical risk and poor prognosis. Under supportive care, peritonitis became progressively evident in the following days. Multiple bacterial floras were isolated from the ascites culture, and blood culture yielded Clostridium bifermentans. Even when broad-spectrum antibiotics were used the clinical condition went downhill gradually. Finally, he died 16 days postresuscitation because of sepsis and multiple organ failure.

3. Discussion

Pneumatosis intestinalis is a clinical condition characterized by gas accumulation in the intestinal wall. It is often a clue rather than a definite diagnosis, suggesting the presence

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Fig. 2  A, Abdominal echogram revealed echogenic gas within the circumference of the ascending colon. This “circle sign” indicated PI of the bowel. B, Abdominal CT 12 hours postresuscitation revealed air within the dependent part of the wall in ascending colon (short arrows). C, The air density can be seen more clearly at lung window (long arrows). D, Progression of the PI (arrowheads) at the same segment of ascending colon was noted 12 days postresuscitation.
of severe intra-abdominal pathologies like necrotizing enterocolitis. In recent years, a number of variant etiologies have been identified, including vasculitis, pulmonary diseases, AIDS, endoscopy-related complications, adverse reaction of medication, and organ transplantation [3]. Several pathogenic mechanisms have also been proposed, such as mechanical theory, with gas intrusion from the gut lumen, and bacterial theory, with gas-forming organisms in, around the gut wall, or within the gut lumen [3,5]. For mechanical theory, intrusion of intraluminal gas into the gut wall might be due to increased intraluminal pressure, enhanced gut permeability because of defects in mucosa or gut’s immune barrier, or a combination of both. As to bacterial theory, the gas within the gut wall might be produced by gas-forming organisms invading the gut wall or by diffusion across the mucosa due to a gradient between intraluminal and serum partial pressure. The gas-forming bacilli, mainly the Clostridium species, are especially likely to overgrow and enter submucosa through the mucosal rents if the gut is ischemic or infarcted.

Hepatic portal venous gas is also a clinical finding suggestive of intra-abdominal pathologies. It might result from bowel necrosis, ulcerative colitis, intra-abdominal abscess, small intestinal obstruction, or gastric ulcers, of which bowel necrosis accounts for more than two thirds of the cases [2]. If bowel ischemic or infarction is the etiology, it is usually regarded as an ominous sign, particularly when associated with metabolic acidosis or elevated serum amylase [6]. As PI and HPVG often implicate poor prognosis if not treated properly, urgent surgical exploration is usually recommended.

For the 2 patients described above, the causes of cardiac arrest were cardiogenic and hypovolemic shock, respectively. Pneumatosis intestinalis and hepatic portal venous gas were considered complications rather than etiologies of cardiac arrests because there were no significant symptoms or signs suggestive of ischemic bowel disease preceding the cardiac arrest events. Besides, PI and HPVG were not clinically evident until the second day postresuscitation. The underlying pathology was most probably severe bowel ischemia followed by necrotizing enterocolitis, which developed after prolonged cardiac arrest and CPR. However, what is the pathogenic mechanism responsible for the bowel infarction after resuscitation? What clinical factors contribute to its occurrence and severity? We herein proposed 2 hypotheses that might explain the development of PI and HPVG after cardiac arrest and CPR.

First, bowel ischemia may develop soon after cardiac arrest because the mesentery perfusion ceased upon global circulatory arrest. This is especially true if CPR is not immediately instituted. Even if the cardiac arrest is witnessed and CPR is carried out promptly, the mesentery perfusion might still be poor because the cardiac output produced by manual external chest compression is only about 20% that of normal [7]. The organ perfusion might be even worse if atherosclerosis of the corresponding vasculature is severe. As shown in the first case, marked atherosclerosis of the abdominal aorta and stenosis of the superior mesentery artery orifice were noted on CT scan (Fig. 1D). Even if the patient had no signs of bowel ischemia under normal condition, the bowel perfusion must be severely compromised after prolonged CPR, which in turn led to severe bowel ischemia and even infarction [8].

In addition to reduced blood flow resulting from preexisting atherosclerosis and vascular stenosis, severe vasoconstriction during and after CPR might further compromise the mesentery perfusion. The vasoconstriction mostly comes from exogenous vasoactive agents administered for resuscitation [9-11], though endogenous angiotensin may also have some roles [12]. The effect of vasoconstriction is evident not only during CPR, but may be more pronounced in the postresuscitation phase because high dose of vasopressors is often used in this period for maintenance of hemodynamics. As a consequence, the duration of vasoconstriction and bowel ischemia is inevitably prolonged, exceeding the tolerated tolerance of the intestine and leading to bowel infarction and necrotizing enterocolitis.

For the 2 patients described above, large doses of epinephrine and vasopressin were administered during prolonged CPR, followed by high-dose dopamine and norepinephrine infusion in the postresuscitation phase. Severe mesentery vasoconstriction with resultant bowel ischemia or infarction was predictable, which might progress to necrotizing enterocolitis and later became clinically detected as PI and HPVG. Moreover, in the presence of severe bowel ischemia with defected mucosal barrier, it was favorable for overgrowth of the gas-forming bacilli and invasion into the submucosal layer. This might also contribute to the development of PI and HPVG. In the second case of this report, the blood culture of C. bifermentans offers some evidence standing for bacterial theory as the pathogenic mechanism of PI and HPVG.

With regard to prognosis, the development of PI and HPVG after CPR seems to proclaim a poor outcome. Thomachot et al [13] in a French literature reported a case of HPVG after transient cardiac arrest and CPR. Diffuse mesenteric ischemia was noted at laparotomy, and the patient died of multiple organ failure in the subsequent hours. The outcomes of the 2 patients in this report were dismal, too. Though the survival was lengthened for a few days, both patients eventually died of multiple organ failure. In contrast to the clinical course of patients with isolated ischemic bowel disease, development of PI and HPVG after cardiac arrest and CPR may be a hallmark of devastating ischemic insult to all visceral organs and serves as an ominous prognostic sign. The role of surgical exploration under such circumstances may be limited.

In conclusion, PI and HPVG may complicate the postresuscitation course in patients receiving prolonged CPR. Decreased mesentery perfusion with bowel ischemia or even necrotizing enterocolitis is the proposed pathogenic mechanism. Though not commonly seen, such complica-
itions should be considered in those with old age and severe atherosclerosis, especially when large dose of vasoconstrictive agents have been administered during and after CPR. Development of PI and HPVG in the postresuscitation phase not only suggests the presence of bowel ischemia, but also signifies poor perfusion of the visceral organs. Progression to multiple organ failure with poor prognosis is often anticipated. Surgical exploration of the abdomen may not be helpful. Ways to eliminate the poor outcomes may be focused more on prevention than on treatment afterward. Improving the visceral organ perfusion by effective CPR techniques and reducing splanchnic vasoconstriction by minimizing vasopressor dosage in the postresuscitation period are the key points for preventing its occurrence.

References

Intubation without premedication may worsen outcome for unconsciousness patients with intracranial hemorrhage

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1. Introduction

Among the first steps of first aid in either the field or in an emergency department (ED), securing the airway is always one of the most important maneuvers, and tracheal intubation is the most reliable method to maintain the airway [1]. In the case of tracheal intubation for intracranial hemorrhage, premedications (sedatives, painkillers, muscle relaxants, and/or depressors of blood pressure) are routinely given to prevent an increase of intracranial hemorrhage due to stress reaction [2]. At our ED, some severely unconscious patients received premedication before intubation; however, others did not receive premedication before intubation, based on the judgment of the physicians on duty. The reasons for not giving premedication are the following: (1) the unconscious patients had not yet been precisely diagnosed and they had received first aid before the diagnosis; (2) using sedatives such as diazepam before intubation tends to make the physical examination for neurological findings extremely difficult; and (3) it remains unknown as to whether intubation without premedication...
for severe unconscious patient is really stressful or not. We thus retrospectively reviewed the medical records of such patients to clarify whether intubation with or without premedication affected the patient’s outcome or not.

2. Methods

From May 1995 to June 2001, 1456 patients were intubated at our ED (Table 1). Among them, 70 patients who demonstrated nontraumatic intracranial hemorrhage by head computed tomography (CT), except for cardiopulmonary arrest on arrival, were investigated as subjects. Every case was transferred to our ED within 60 minutes from the first emergency call and intubated within 15 minutes from arrival. They were divided into 2 groups, consisting of a drug group (n = 15), wherein drugs were used before intubation, and a control group (n = 55), wherein no drugs were used before intubation. The physical findings (Glasgow Coma Scale [GCS], systolic blood pressure, and pupillary reaction to light) on admission, the head CT findings (subarachnoid or intracerebral hemorrhage), and the Glasgow Outcome Score (GOS) at 3 months from admission were analyzed between the groups by reviewing the medical chart. Regarding the head CT findings, both Fisher’s classification [3] for subarachnoid hemorrhage and the location for intracerebral hemorrhages were investigated. The subgroups consisting of both the subarachnoid and intracerebral hemorrhage groups were divided based on the head CT findings and were analyzed regarding the patients’ background and the GOS. The $\chi^2$ test, Student t test, and Mann-Whitney U test were used for the statistical analyses. A $P$ value less than .05 was considered significant.

3. Results

In the drug group, diazepam (10 mg, n = 9), pentazocine (15 mg, n = 4), fentanyl citrate (0.1 mg, n = 1), lidocaine (50 mg, n = 1), nifedipine (5 mg, n = 6), and nicardipine (2 mg, n = 2) were used before tracheal intubation. Diazepam-pentazocine (n = 3), diazepam-pentazocine-nifedipine (n = 1), diazepam-lidocaine (n = 1), diazepam-nicardipine (n = 1), and nifedipine-nicardipine (n = 1) were used in combination based on the decision of the physicians on duty. The backgrounds of the subjects are shown in Table 2. There were no significant changes regarding the sex ratio, age, head CT findings, GCS, systolic blood pressure, and the existence of a light reflex between the 2 groups. In the control group, high blood pressure was not controlled before the examination of head CT. Neither Fisher’s classification for the GOS at 3 months from admission (n = 70) was considered significant.

Table 1 Diagnosis of incubated patients (n = 1456)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>n (%)</th>
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<tbody>
<tr>
<td>Cardiopulmonary arrest (including trauma)</td>
<td>895 (61.5)</td>
</tr>
<tr>
<td>Trauma</td>
<td>199 (13.6)</td>
</tr>
<tr>
<td>Drug intoxication</td>
<td>119 (8.1)</td>
</tr>
<tr>
<td>Stroke (including 7 cases of cerebral infarction)</td>
<td>77 (5.2)</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>41 (2.9)</td>
</tr>
<tr>
<td>Burn</td>
<td>36 (2.5)</td>
</tr>
<tr>
<td>Cardiac diseases including arrhythmia</td>
<td>25 (1.8)</td>
</tr>
<tr>
<td>Neurogenic diseases except stroke</td>
<td>23 (1.6)</td>
</tr>
<tr>
<td>Shock</td>
<td>14 (1.0)</td>
</tr>
<tr>
<td>Metabolic diseases</td>
<td>10 (0.7)</td>
</tr>
<tr>
<td>Others</td>
<td>17 (1.1)</td>
</tr>
<tr>
<td>Total</td>
<td>1456 (100.0)</td>
</tr>
</tbody>
</table>

Table 2 Background of subjects (n = 70)

<table>
<thead>
<tr>
<th></th>
<th>Drug group</th>
<th>Control group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>6 (40.0)</td>
<td>26 (47.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Female</td>
<td>9 (60.0)</td>
<td>29 (52.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Age</td>
<td>57.8 ± 3.8</td>
<td>61.7 ± 1.9</td>
<td>NS</td>
</tr>
<tr>
<td>Disease of</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>intracranial hemorrhage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>9 (60.0)</td>
<td>34 (61.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>6 (41.7)</td>
<td>21 (38.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Initial state on arrival</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GCS</td>
<td>4.6 ± 0.3</td>
<td>4.3 ± 0.1</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>162.4 ± 16.2</td>
<td>171.4 ± 7.8</td>
<td>NS</td>
</tr>
<tr>
<td>Reactive pupil (number)</td>
<td>6 (40.0)</td>
<td>17 (30.9)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are given as n (%) or mean ± SE. NS indicates not significant.

Table 3 CT classification of subjects

<table>
<thead>
<tr>
<th></th>
<th>Drug group</th>
<th>Control group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fisher’s classification of subarachnoid hemorrhage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>n = 9</td>
<td>n = 34</td>
<td>NS</td>
</tr>
<tr>
<td>Group 3</td>
<td>0</td>
<td>1 (2.9)</td>
<td></td>
</tr>
<tr>
<td>Group 4</td>
<td>8 (88.8)</td>
<td>19 (55.8)</td>
<td></td>
</tr>
<tr>
<td>Location of intracerebral hemorrhage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Putamen and/or thalamus</td>
<td>3 (50.0)</td>
<td>9 (42.9)</td>
<td></td>
</tr>
<tr>
<td>Lobar</td>
<td>1 (16.6)</td>
<td>1 (4.7)</td>
<td></td>
</tr>
<tr>
<td>Brainstem</td>
<td>2 (34.4)</td>
<td>8 (38.1)</td>
<td></td>
</tr>
<tr>
<td>Cerebellum</td>
<td>0</td>
<td>1 (4.7)</td>
<td></td>
</tr>
<tr>
<td>Intraventricular hemorrhage</td>
<td>0</td>
<td>2 (9.6)</td>
<td></td>
</tr>
</tbody>
</table>

Values are given as n (%). NS indicates not significant.

Table 4 GOS at 3 months from admission (n = 70)

<table>
<thead>
<tr>
<th></th>
<th>Score</th>
<th>Drug group</th>
<th>Control group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good recovery</td>
<td>1</td>
<td>6 (40.0)</td>
<td>2 (3.6)</td>
<td>.001</td>
</tr>
<tr>
<td>Moderate</td>
<td>2</td>
<td>1 (6.6)</td>
<td>3 (5.4)</td>
<td></td>
</tr>
<tr>
<td>disability</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe disability</td>
<td>3</td>
<td>0</td>
<td>3 (5.4)</td>
<td></td>
</tr>
<tr>
<td>Vegetative state</td>
<td>4</td>
<td>1 (6.6)</td>
<td>7 (12.7)</td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>5</td>
<td>7 (46.8)</td>
<td>40 (72.9)</td>
<td></td>
</tr>
<tr>
<td>Average of GOS</td>
<td>3.1 ± 0.5</td>
<td>4.4 ± 0.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are given as n (%) or mean ± SE.
Table 5  Background and outcome of patients with intracerebral hemorrhages (n = 27)

<table>
<thead>
<tr>
<th></th>
<th>Drug group (n = 6)</th>
<th>Control group (n = 21)</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3 (50.0)</td>
<td>15 (71.4)</td>
<td>NS</td>
</tr>
<tr>
<td>Female</td>
<td>3 (50.0)</td>
<td>6 (29.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Age</td>
<td>53.3 ± 8.4</td>
<td>58.1 ± 3.7</td>
<td>NS</td>
</tr>
<tr>
<td>GCS</td>
<td>3.8 ± 0.3</td>
<td>4.5 ± 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>204.8 ± 30.2</td>
<td>183.4 ± 11.0</td>
<td>NS</td>
</tr>
<tr>
<td>Reactive pupil</td>
<td>1 (16.6)</td>
<td>5 (23.8)</td>
<td>NS</td>
</tr>
<tr>
<td>GOS</td>
<td>4.1 ± 0.6</td>
<td>4.6 ± 0.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are given as n (%) or mean ± SE. NS indicates not significant.

subarachnoid hemorrhage group nor the location of the intracerebral hemorrhage group showed any significant change between the 2 groups (Table 3). The average GOS in the drug group (3.1 ± 0.5) was significantly smaller than in the control group (Table 4). The reason for death in all subjects was brain death. Based on a review of the medical chart, 13 cases showed a deterioration of symptoms (loss of light reflex, n = 6, decrease of GCS, n = 4, respiratory arrest, n = 3) in the control group, but none in the drug group.

Regarding both the subarachnoid and intracerebral hemorrhage group, no significant change was observed regarding the background (Tables 5 and 6). In the subarachnoid groups, the average GOS in the drug group (2.4 ± 0.6) was significant smaller than in the control group. In the intracerebral hemorrhage group, the average GOS in the drug group (4.1 ± 0.6) was smaller than in the control group (4.6 ± 0.1), however, the difference was not significant.

4. Discussion

Our results indicated that in the case of intubation for unconscious patients who may have intracranial hemorrhage, especially in the subarachnoid space, the lack of premedication before intubation was associated with a worse outcome.

Tracheal intubation without premedication may be accompanied by increased blood pressure because of a stress reaction or bucking reflex [2,4]. The higher the increase in blood pressure, the higher the increase in intracranial hemorrhage [5-7]. In our study, insufficient blood pressure records made it difficult to identify a higher increase of blood pressure associated with no premedication before intubation than with premedication. In addition, the head CT findings after intubation did not demonstrate any change between the groups. However, almost all CT head examinations were performed within 30 minutes from intubation in our department. As a result, the rapid examinations may have failed to demonstrate rebleeding or increasing of hemorrhage induced by intubation. Furthermore, there were 13 cases that deteriorated immediately after intubation in the control group (which meant rebleeding or increase in intracranial hemorrhage), but no such cases were found in the drug group. Accordingly, if a patient with intracranial hemorrhage shows severe unconsciousness and feels no pain, then intubation without premedication may cause an increased blood pressure, thereby leading to an increase in the occurrence of intracranial hemorrhage and a worse outcome.

Several problems exist in our study. First, although there was no statistically significant difference between the 2 groups, patients in the drug group were younger, had a higher percentage of reactive pupils, and classified as group 4 in Fisher’s classification. It has been shown that age, pupillary reaction, and hemorrhage volume are important prognostic factors [8-11]. Because of the retrospective nature of this analysis, it was hard to exclude this variance completely.

Second, there are many diseases that cause unconsciousness; thus, in an undiagnosed intracranial hemorrhage in the field or at an ED before a CT head examination, premedication may lead to life-threatening consequences [12,13]. Even a suspicious diagnosis can turn out to be correct in the end; therefore, premedication with intubation before the identification of any other associated diseases [14,15] may also cause life-threatening problems.

Third, in cerebral vascular diseases, cerebral infarction requires a high blood pressure to save the ischemic penumbra area [16], whereas an intracranial hemorrhage needs a lower blood pressure to avoid any increase in size. Clinically, it is very difficult to discern a cerebral hemorrhage from infarction in unconscious patients without a head CT examination. As a result, the misuse of blood pressure depressor drugs for cerebral infarction with unconsciousness may increase the size of the cerebral infarct and lead to a deteriorated outcome.

Fourth, most of the drugs used in the drug group are not typical premedications for tracheal intubation in the operating room [2]. If the drugs are similar to those commonly used in the operating room, then such patients may have an even better outcome. Finally, the drugs or combinations of drugs that were most beneficial to the

Table 6  Background and outcome of patients with subarachnoid hemorrhages (n = 25)

<table>
<thead>
<tr>
<th></th>
<th>Drug group (n = 9)</th>
<th>Control group (n = 34)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3 (33.3)</td>
<td>11 (32.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Female</td>
<td>6 (66.7)</td>
<td>23 (68.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Age</td>
<td>60.8 ± 3.1</td>
<td>63.9 ± 2.0</td>
<td>NS</td>
</tr>
<tr>
<td>GCS</td>
<td>5.2 ± 0.5</td>
<td>4.2 ± 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>134.2 ± 11.8</td>
<td>163.6 ± 10.7</td>
<td>NS</td>
</tr>
<tr>
<td>Reactive pupil</td>
<td>5 (55.5)</td>
<td>12 (35.2)</td>
<td>NS</td>
</tr>
<tr>
<td>GOS</td>
<td>2.2 ± 1.8</td>
<td>4.3 ± 1.3</td>
<td>.001</td>
</tr>
</tbody>
</table>

Values are given as n (%) or mean ± SE. NS indicates not significant.
patient before intubation were not clarified in this retrospective study. To elucidate these drugs, further human investigations are warranted.

References


Dental fracture risk of metal vs plastic laryngoscope blades in dental models

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Received 29 March 2004; accepted 29 March 2004

Abstract

Background: Dental fracture is a complication of laryngoscopy and endotracheal intubation.
Purpose: The purpose of this study is to compare the potential fracture rates in dental models using metal laryngoscope blades with those using plastic laryngoscope blades.
Methods: Size 3 Macintosh plastic and metal laryngoscope blades were applied against 4 different dental model materials at varying torques to determine when the dental model material would fracture.
Results: The plastic blade did not fracture any of the dental model materials. The metal blade fractured the glass dental model material even at the lowest torque setting. At a moderate torque setting, the plastic blade showed evidence of structural failure, whereas the metal blade did not fail at any torque setting. Fracture of wooden dowel and ceramic teeth model materials occurred with the metal blade but only at torque settings higher than what the plastic blade could achieve.
Conclusion: Based on the dental fracture models studied, plastic laryngoscope blades have a lower potential for dental fracture compared with metal blades. Plastic laryngoscope blades would be best suited for trainees performing routine intubations under direct supervision. Metal blades would be more advantageous in difficult intubations preferably done by experienced intubators.

1. Introduction

Dental fracture is a common complication of tracheal intubation [1]. In a study of 366 patients by McGovern et al [1], dental fracture was the most common complication. A retrospective study of 598,904 consecutive patients requiring anesthesia over an 11-year period found the dental injury rate to be as high as 1 in 4 patients [2]. These injuries not only have a direct comfort and cosmetic effect but may also increase the risk of aspiration. Repair of dental injuries is significant and can amount to US$782 per incident [2]. Despite their availability, plastic laryngoscope blades are not routinely used in residency programs for intubations by physicians in training.

Although plastic blades may reduce the risk of dental fractures, no study has compared plastic laryngoscope...
blades with metal blades. The purpose of this study is to compare the potential fracture rates in dental models using metal laryngoscope blades and those using plastic laryngoscope blades.

2. Methods

A standard #3 Macintosh stainless steel laryngoscope blade was attached to a standard laryngoscope handle. Plastic #3 curved Macintosh laryngoscope blades were obtained in 3 variants and from 3 sources: (1) Rusch Lite blade kit (Rusch, Duluth, Ga, purchased from Moore Medical, New Britain, Conn), (2) PMX Medical disposable laryngoscope blades (PMX Medical, Salt Lake City, Utah), and (3) Vital View laryngoscope blades (Vital Signs Inc, Totowa, NJ).

Fig. 1 describes the study model. Various dental model materials were placed in a vise and oriented 4 cm from the base of the blade. The dental model materials were oriented perpendicular and tangent to the side adjacent the palate-facing surface of the laryngoscope blade (at the “fulcrum” position). The tip of the laryngoscope blade was held in a fixed position. A preset torque was set on a torque wrench (Mac Tools TWX80, Columbus, Ohio) at 5, 10, 15, 20, 25, and 30 ft-lb (1 ft-lb = 1.355 Nm). A rotational force was applied to the handle of the laryngoscope apparatus via a torque wrench. Rotation was ceased if (1) the preset torque was met, (2) the dental model was compromised, or (3) the laryngoscope blade was compromised. Compromise of the dental model was defined as any visible/audible break, chip, or crack in the dental model. Compromise of the laryngoscope blade was defined as any visible/audible break, crack, chip, or deformation of the blade.

Various dental model materials were placed in the vise holding the “dental model.” These included ceramic teeth (A20A-200 ceramic composite crown with epoxy dentin upper anterior incisors, Kilgore International, Coldwater, Mich), a 3.2 cm diameter by 2.5 cm long hardwood dowel, a 2.5 × 0.32 cm ceramic tile (#D3141MFIP, Daltile Corp, Dallas, Tex), and a 1.3 × 1.3 × 0.16 cm glass piece (#1613-49, Darice Inc, Strongville, Ohio). The ceramic tile and the glass dental models were oriented in a vise so that their planes were perpendicular to the plane of the laryngoscope blades. The ceramic tile protruded 1.2 cm above the vise with the remaining 1.3 cm within the vise. The glass piece protruded 0.6 cm above the vise with the remaining 0.7 cm within the vise. The ceramic tooth was held in an approximate anatomic position. The base of the tooth was 1.3 cm within the vise and protruded 1.2 cm in above the vise. The 3.2 mm wooden dowel was 1.4 cm within the vise and protruded 1.2 cm outside the vise.

Five trials with each dental model material type with both laryngoscope blade types were run at multiple torque wrench settings. The torque wrench was initially set at a torque of 5 ft-lb (6.8 Nm). The torque was increased by an interval of 5 ft-lb until a maximum of 40 ft-lb was achieved. The respective preset torque intervals at which either the blades or the dental models were compromised were recorded and compared.

3. Results

The results are summarized in Table 1. Plastic laryngoscope blades failed to fracture any of the dental model materials in all torque ranges. The plastic blade integrity was compromised at the 11 to 15 ft-lb range. Thus, higher torque
ranges could not be attempted. This essentially functions as a “stop level,” such that the application of forces above this level is not possible (similar to a “pop-off valve” during handbag ventilation).

The metal blade fractured all the glass model materials even at the lowest torque range compared with no fractures with the plastic blade (P < .05). The metal blade began to fracture the wooden dowel at the 11 to 15 ft-lb range, and by the 21 to 25 ft-lb range, 100% were fractured. The metal blade fractured all the ceramic teeth at the 16 to 20 ft-lb range. None of the metal Macintosh blades experienced structural compromise.

In earlier preliminary trials with straight blades, a metal #2 straight blade experienced structural compromise at a high torque level with the ceramic tile. The blade was too expensive to be repeatedly replaced, so the study was carried out using only curved Macintosh blades. Similarly, the plastic straight blades that were used for the straight blade comparisons would bend at lower torques compared with the curved plastic blades. Metal laryngoscope blades did not suffer any damage at any torque interval.

### 4. Discussion

There are no present studies that compare dental injury outcomes of metal vs plastic laryngoscope blades used in intubation. This study demonstrates that plastic laryngoscope blades consistently break at a torque interval of 11 to 15 ft-lb whereas metal blades do not, regardless of the dental model used (ie, ceramic tile, wood, glass, ceramic teeth) (P < .05). The glass model suggests that the plastic blade has a lower risk of dental fracture because the metal blade fractured the glass in all the trials even at the lowest torque setting whereas the plastic blade was not able to fracture the glass even at a higher torque setting.

In addition, the maximum torque that the plastic blade can apply is limited by its own structural composition. Because the metal blade is much stronger, the maximum torque of the metal blade is essentially unlimited within the range of normal human strength.

The torque required for normal intubation ranges between 2.7 and 10.8 ft-lb (2-8 Nm) [3]. The plastic blade is strong enough to achieve this torque through most of this torque range, but at the upper limit of this range, the plastic blade integrity might be compromised. It is difficult to be certain of this because the concept of “torque” in this study might not be exactly identical to the torque measurements obtained in our study.

The validity of these dental fracture models has not been proven. Different model materials were used to simulate a variety and range of dental strength characteristics that might be encountered. Human dentition strength is variable, ranging from weak to very strong. Using extracted human teeth would have introduced structural strength variation between the different trials. It would have been potentially misleading to use a small number of teeth of variable structural strength. Such “variance” within the sample itself could only be overcome to reveal the true difference only if a large number of human teeth were to be used (not easily obtained and not feasible). Thus, the only practical and ethical alternative to study this problem is to use models of different types that have some structural resemblance to human teeth.

During the process of endotracheal intubation, laryngoscopy places the distal end of the laryngoscope blade in the vallecula. Although the term “torque” is used in our study and by others, there should ideally be no torque applied to the teeth. Using the teeth as a fulcrum is a common mechanism of dental fracture. The laryngoscope blade has a distal side opposite the laryngoscope blade (facing the vallecula).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Dental model fracture results with increasing torque</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model material</td>
<td>Torque range (in ft-lb)</td>
</tr>
<tr>
<td>Wooden dowel</td>
<td>Metal blades</td>
</tr>
<tr>
<td></td>
<td>Plastic blades</td>
</tr>
<tr>
<td>Ceramic tile</td>
<td>Metal blades</td>
</tr>
<tr>
<td></td>
<td>Plastic blades</td>
</tr>
<tr>
<td>Glass</td>
<td>Metal blades</td>
</tr>
<tr>
<td></td>
<td>Plastic blades</td>
</tr>
<tr>
<td>Ceramic teeth</td>
<td>Metal blades</td>
</tr>
<tr>
<td></td>
<td>Plastic blades</td>
</tr>
</tbody>
</table>

Percent of 5 trials in which model material compromise was sustained. n/a indicates that the plastic laryngoscope blade was compromised before any dental model material was compromised.

\(^a\) Represents statistical significance where dental models in the plastic blade trial broke whereas models in the metal blade trial did not break at given torque intervals, P < .05.
palate), which has the potential of fulcrum against the upper teeth, and has a proximal side closer to the laryngoscope handle (facing the tongue), which has the potential (but less likely) to fulcrum against the lower teeth. Our model only tested the dental fracture risk associated with the distal side (palate side) of the laryngoscope blade, where it potentially impacts on the upper teeth.

Although experienced emergency physicians are comfortable with most intubations using the standard metal blades, many emergency physicians must also supervise endotracheal intubation done by physicians in training. The difficulties of supervising this procedure include the critical nature of a patient’s condition and the inability to see what the intubator sees. At an angle, the supervising attending physician cannot see the vocal cords. In addition, if a trainee decides to descend the laryngoscope blade against a patient’s upper teeth and torque the blade, this could happen too fast for the supervising attending physician to prevent.

Our study suggests that plastic laryngoscope blades have a lower dental fracture potential. This would be very useful in the supervision of endotracheal intubation by less-experienced trainees. If a trainee were to torque the blade against a patient’s teeth as a fulcrum, it would be less likely to cause a dental fracture. This would give the supervising physician the opportunity to correct the trainee’s technique without letting the patient sustain any dental fracture. This would permit the supervising physician to focus more attention on securing the airway.

During these trials, the plastic blades were noted to have an audible click, without gross permanent deformation of the blade. The audible click could serve as a warning for a trainee intubator. Thus, a trainee using the plastic blade could potentially hear an audible click as a warning that a high torque has been applied and that the blade has sustained a crack somewhere within it. The blade could potentially break, resulting in an intraoral injury. Thus, the blade should be removed as soon as any sign (audible, visual, or by feel) that the structural limit of the plastic blade has been reached is obtained.

Difficult intubations require more force applied to the laryngoscope blade [3], in which case, a plastic laryngoscope blade might not be strong enough. Although our study was not designed to demonstrate the maximum torque required for a difficult intubation, our study did demonstrate that metal laryngoscope blades are stronger than plastic blades. Thus, it makes sense to use a metal blade for a potentially difficult intubation. For such difficult intubations, it would be best to have the intubation performed by the most skilled intubator in the room. It would be unwise for a junior-level trainee to attempt such an intubation until a greater experience level is achieved with routine intubations. Thus, plastic laryngoscope blades are more suitable for trainees performing routine intubations under direct supervision.

In summary, based on the dental fracture models studied, plastic laryngoscope blades have a lower potential for dental fracture compared with metal blades. Plastic laryngoscope blades would be best suited for trainees performing routine intubations under direct supervision. For such difficult intubations, it would be best to have the intubation performed by the most skilled intubator in the room. It would be unwise for a junior-level trainee to attempt such an intubation until a greater experience level is achieved with routine intubations. Thus, plastic laryngoscope blades are more suitable for trainees performing routine intubations under direct supervision. Metal blades would be more advantageous in difficult intubations preferably done by experienced intubators.

References

Propofol for deep procedural sedation in the ED

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Received 19 March 2004; accepted 25 May 2004

Abstract
We sought to evaluate the use of propofol (2,6-diisopropylphenol) for ED procedural sedation, particularly when administered in a routine fashion for a variety of indications.

Methods: This was a prospective observational study conducted in an urban teaching ED. Propofol was administered by handheld syringe and combined with fentanyl. Measurements included propofol and fentanyl dose, serial vital signs, pulse oximetry, adverse events, and patient and physician satisfaction.

Results: One hundred thirty-six subjects (18 to 69 years) were enrolled. Procedures included 82 (60.3%) abscess incision and drainages and 47 (34.6%) orthopedic reductions. Adverse events occurred in 14 cases (10.3%; 95% confidence interval 5.2% to 15.4%), including hypotension in 5, hypoxemia in 7, and apnea in 5. One patient required intubation. Both patient and physician satisfaction were excellent.

Conclusions: ED procedural sedation with propofol was effective and well accepted by patients and physicians. However, it produced a significant incidence of hypotension, hypoxemia, and apnea.

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1. Introduction

Patients undergoing painful procedures in the ED frequently require a potent sedative, in addition to narcotic analgesia. Propofol (2,6-diisopropylphenol) offers an alternative to other sedative agents such as midazolam, methohexital, and ketamine. Advantages of propofol include its very rapid onset of action and short duration of action as well as its antiemetic and amnestic properties.

Problems associated with propofol include apnea, hypotension, loss of protective reflexes, and a propensity to induce rapid swings between light sedation and general anesthesia. The potential danger of using propofol in the ED has been compared to driving a turbo-charged sports car, and editorialists have pointed out the need for a large prospective case series evaluating the safety and efficacy of routine propofol use by emergency physicians \[1,2\]. To date, there have been 8 published studies that specifically address the use of propofol in the ED setting \[3-10\]. Most of these studies have been conducted in children and under the closely controlled conditions of a comparative trial.

We designed a prospective observational study to evaluate the safety and efficacy of propofol for deep procedural sedation (PS) in the ED. Our primary objective...
was to measure and record in detail the adverse reactions associated with propofol, particularly when administered by handheld syringe and used in a routine fashion for a broad range of procedures in a busy teaching ED. We sought to record and describe in detail the dose and rate of administration that was required. In addition, patient and provider satisfaction was assessed.

2. Methods

2.1. Study design

This study was a prospective observational study. The institutional review board of Alameda County Medical Center–Highland Hospital approved the study protocol.

2.2. Study setting and population

The study was performed in the ED of an urban county hospital with an annual census of 85,000. All patients ≥18 years requiring sedation for a painful procedure were eligible. Exclusion criteria were hypotension, a known allergy to eggs or soybeans, or pregnancy. Propofol was administered by ED attending physicians and senior residents, including but not limited to the authors of the study. These providers were asked to review and follow guidelines written at the top of every study data collection sheet but otherwise underwent no special training in the use of propofol.

2.3. Study protocol

All patients enrolled in the study underwent PS in adherence with a standard ED protocol. Intravenous (IV) access was established. Supplemental oxygen was administered via nasal cannula or bag-valve-mask (BVM). Patients were required to be NPO for 2 hours for liquids and for 6 hours for solids. Patients underwent continuous cardiac, pulse oximetry, and automated blood pressure monitoring with resuscitation equipment at the bedside. A designated PS nurse was required to be in attendance. Providers who administered propofol were dedicated strictly to providing PS and were not involved in performing the procedure itself.

Guidelines for administration of fentanyl and propofol were written at the top of each study data sheet. It was recommended that pretreatment with fentanyl be given as a 1 to 1.5 μg/kg bolus 1 minute before administration of propofol. Repeat fentanyl doses were given at the discretion of the provider. Propofol (10 mg/mL) was delivered by hand from a 10-mL syringe. Two 10-mL syringes were filled with propofol before beginning. It was recommended that an initial propofol bolus of 0.5 to 1.0 mg/kg be given over 30 seconds. Half the normal initial bolus dose was recommended in patients older than 65. Propofol was then infused by hand and titrated to a level of sedation deemed adequate for the procedure. The recommended technique for propofol infusion was to give 0.1 to 0.2 mg/kg as an intermittent microbolus every 30 to 60 seconds as needed. There was no recommended maximum total propofol dose.

2.4. Measurements

Medication doses, administration times, and total procedure time were recorded. Patient weights mostly were based on the patient’s own estimate. All side effects and adverse reactions were recorded. Hypotension was defined as a drop in systolic blood pressure to less than 90 mm Hg. Hypoxemia was defined as oxygen saturation less than 90%. Apnea was defined as absence of spontaneous ventilation lasting 30 seconds. After the procedure, the physician providing PS was asked to rate their satisfaction with propofol as excellent, satisfactory, or unsatisfactory.

A table is included with data on adverse events and interventions. The table includes columns for age/sex, procedure/min, fentanyl bolus (μg/kg), total fentanyl (μg/kg), propofol initial bolus (mg/kg), titrated dose (mg/kg/min), complication, intervention, and outcome. The table also includes abbreviations for Asp, AWM, BVM, I&D, IVF, O2, and %.

Table 1 Analysis of adverse events

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>Procedure/min</th>
<th>Fentanyl</th>
<th>Propofol</th>
<th>Complication</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>18/M</td>
<td>Reduction/5</td>
<td>0.8</td>
<td>1.5</td>
<td>0.8</td>
<td>0.12</td>
<td>Apnea; desaturation 75%; emesis</td>
</tr>
<tr>
<td>56/M</td>
<td>Reduction/10</td>
<td>None</td>
<td>none</td>
<td>1</td>
<td>0.1</td>
<td>Desaturation 87%</td>
</tr>
<tr>
<td>25/M</td>
<td>Reduction/5</td>
<td>1.1</td>
<td>1.1</td>
<td>1.5</td>
<td>0.27</td>
<td>Desaturation 86%</td>
</tr>
<tr>
<td>18/M</td>
<td>Reduction/1</td>
<td>1.4</td>
<td>1.4</td>
<td>1.4</td>
<td>0.41</td>
<td>Desaturation 84%</td>
</tr>
<tr>
<td>32/F</td>
<td>I&amp;D/2</td>
<td>2.7</td>
<td>2.7</td>
<td>1.4</td>
<td>0.11</td>
<td>Apnea; desaturation 80%</td>
</tr>
<tr>
<td>35/M</td>
<td>Penis asp/5</td>
<td>1.1</td>
<td>2.2</td>
<td>1.1</td>
<td>0.11</td>
<td>Apnea; desaturation 89%</td>
</tr>
<tr>
<td>22/M</td>
<td>Reduction/5</td>
<td>1.4</td>
<td>1.4</td>
<td>1.4</td>
<td>0.14</td>
<td>Apnea; no desaturation</td>
</tr>
<tr>
<td>40/M</td>
<td>Reduction/0.3</td>
<td>2.6</td>
<td>2.6</td>
<td>1</td>
<td>None</td>
<td>Apnea; no desaturation</td>
</tr>
<tr>
<td>31/M</td>
<td>Reduction/4</td>
<td>0.7</td>
<td>0.7</td>
<td>0.9</td>
<td>0.43</td>
<td>Apnea; no desaturation</td>
</tr>
<tr>
<td>39/M</td>
<td>Reduction/5</td>
<td>1.6</td>
<td>4.1</td>
<td>1.3</td>
<td>0.13</td>
<td>Hypotension 89/45</td>
</tr>
<tr>
<td>22/M</td>
<td>I&amp;D/7</td>
<td>0.8</td>
<td>1.6</td>
<td>1</td>
<td>0.41</td>
<td>Hypotension 74/49</td>
</tr>
<tr>
<td>56/M</td>
<td>I&amp;D/9</td>
<td>1.5</td>
<td>1.5</td>
<td>0.9</td>
<td>0.14</td>
<td>Hypotension 83/59</td>
</tr>
<tr>
<td>24/F</td>
<td>I&amp;D/3</td>
<td>1.8</td>
<td>1.8</td>
<td>1.8</td>
<td>0.58</td>
<td>Hypotension 80/40</td>
</tr>
<tr>
<td>46/M</td>
<td>I&amp;D/1</td>
<td>None</td>
<td>0.8</td>
<td>None</td>
<td>None</td>
<td>Hypotension 82/40</td>
</tr>
</tbody>
</table>

Abbreviations: Asp indicates aspiration; AWM, airway manipulation; BVM, bag-valve-mask ventilation; I&D, abscess incision and drainage; IVF, IV fluid.
and whether they would use propofol again. Patients answered whether they remembered the procedure and whether they would receive propofol again and rated their pain as none, minimal, moderate, or severe. Study data were recorded prospectively on study data sheets by the physician providing PS and the PS nurse. Missing data were retrieved from PS nursing flow sheets.

2.5. Data analysis

Study data were entered into an Excel (Microsoft Corp, Redmond, Wash) database. Data such as medication dose, infusion rate, and procedure time were expressed as a mean and range. Measured outcomes such as adverse-event rates were expressed as a percent of total cases, accompanied by a 95% confidence interval (CI). Differences in mean medication dosage between cases with and without an adverse event were compared, and the statistical significance was determined by 1-sided \( t \) test. Statistical analyses were performed using SPSS statistical software (SPSS Inc, Chicago, Ill).

3. Results

Over 24 months, a total of 136 subjects were enrolled. Patient age ranged from 18 to 69 years. Procedures included 82 (60.3%) abscess incision and drainages and 47 (34.6%) orthopedic reductions. The remaining 7 were priapism aspiration, wound debridement, tube thoracostomy, diagnostic peritoneal lavage, colostomy prolapse reduction, rectal prolapse reduction, and esophagogastroduodenoscopy. Average procedure time was 8.6 minutes (range 0.22-53 minutes).

The mean initial bolus dose of fentanyl was 1.2 \( \mu g/kg \) (range 0.0-3.9 \( \mu g/kg \)). The mean total fentanyl dose was 2.0 \( \mu g/kg \) (range 0.5-5.8 \( \mu g/kg \)). The mean initial bolus dose of propofol was 0.98 mg/kg (range 0.33-2.00 mg/kg). The mean propofol infusion rate (delivered by intermittent microbolus after the initial bolus dose) was 0.22 mg/kg/min (range 0.02-0.8 mg/kg/min). The mean total propofol dose was 168.6 mg (range 50-540 mg).

Adverse events occurred in a total of 14 (10.3%; 95% CI 5.2%-15.4%) cases. Hypotension occurred in 5 (3.7%; 95% CI 0.5%-6.9%) patients, hypoxemia in 7 (5.1%; 95% CI 1.4%-8.8%), and apnea in 5 (3.7%; 95% CI 0.5%-6.9%). Refer to Table 1 for details.

In a post hoc analysis, we compared the difference in medication dosage between cases in which an adverse event occurred and those without complication. The results of this analysis are summarized in Table 2. We found that, on average, the fentanyl bolus dose, initial propofol bolus dose, and propofol infusion rate were all slightly higher in the group experiencing adverse events. However, only the difference in initial propofol bolus dose was statistically significant.

Physician and patient satisfaction scores are listed in Table 3.

4. Discussion

Propofol (2,6-diisopropylphenol) is an IV sedative-hypnotic agent for use in the induction and maintenance of...
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anesthesia or sedation. Propofol has many properties that make it an attractive agent for PS in the outpatient and ED setting. Propofol has a rapid onset of action, producing hypnosis usually within 40 seconds from the time of injection. Peak effect occurs at 92 seconds [11,12]. Propofol has an ultrashort half-life (distribution t1/2 2-4 minutes) with injection. Peak effect occurs at 92 seconds [11,12]. Propofol has a rapid onset of action, producing hypnosis usually within 40 seconds from the time of injection. Peak effect occurs at 92 seconds [11,12]. Propofol has an ultrashort half-life (distribution t1/2 2-4 minutes) with recovery times of within 5 to 15 minutes [11]. Propofol also has antiemetic properties and is rarely associated with emesis [1]. Disadvantages of propofol include respiratory and hemodynamic depression, narrow therapeutic window, lack of analgesic effect, and lack of a reversal agent [1,11,12].

Since its introduction in 1977 as a general anesthesia induction agent, propofol has gained popularity for PS in many settings. Its use by nonanesthesiologists has been studied and declared safe in the setting of bronchoscopy, cardioversion, percutaneous transluminal coronary angioplasty, endoscopy, and dental procedures [13-17]. To date, there have been 8 published studies specifically examining use of propofol for PS in the ED setting. The largest of these, by Bassett et al [9], involved 393 children who received propofol for brief orthopedic procedures. There have been 4 studies involving adult patients. In one study, propofol was administered by automatic pump to 20 patients [10], and in another, involving 21 patients, the dosing method was not described [6]. In a study by Coll-Vinent et al [8], propofol was administered as a single 1.5 mg/kg bolus to 9 adults undergoing electrical cardioversion. Miner et al [5] described the use of propofol, given as a 1 mg/kg bolus followed by 0.5 mg/kg every 2 minutes as needed, in 51 adult patients undergoing orthopedic procedures. In the study by Miner et al, propofol was administered in the closely controlled setting of a randomized comparative trial with methohexital. Ours is the largest, prospective, ED study of the use of propofol for PS in adults. We sought to measure and characterize adverse events during PS with propofol, particularly when used in a routine fashion in a busy academic ED.

Hypotension is a known dose-dependent complication of propofol that appears to be caused by both vasodilation and myocardial depression. In our study, hypotension occurred in 5 patients (3.7%). (Refer to Table 1 for details.) This was transient, lasting less than 1 minute in every case, yet clinically significant in that it required an IV fluid bolus in each case. Whereas some prior ED propofol studies have reported no significant hypotension [5,8], others found a similar incidence of transient hypotension to our study. In the report by Swanson et al [10], 1 of 20 adults (5%) had a fall in systolic blood pressure to 80 mm Hg that resolved spontaneously by the next blood pressure measurement. Skokan et al [4] found that systolic and diastolic blood pressure fell in all 40 children receiving propofol, requiring fluid bolus in 2. Similarly, Bassett et al [9] found that, among 393 pediatric cases, blood pressure fell transiently in 92% (mean systolic decrease 10 mm Hg). Taken together with these other reports, our findings point out the importance of limiting use of propofol to patients with normal baseline blood pressure and robust cardiovascular reserve.

Respiratory depression from propofol, leading to apnea and/or hypoxemia, appears to be related to dose and rate of administration, as well as to concomitant use of other medications [11,12]. In one ED study, respiratory depression, as detected by end-tidal CO₂ monitoring, occurred in 45% of adults receiving propofol [5]. Hypoxemia (oxygen saturation <90%) was reported to occur in 5% to 44% of patients in prior ED studies [3,4,8,10], although routine use of supplemental oxygen varied. In our study, despite supplemental oxygen, 7 (5.1%) patients experienced hypoxemia, and in 3 cases, this was associated with apnea. One patient, described in detail below, required BVM ventilation followed by endotracheal intubation. In the other 6 patients, hypoxemia was transient and resolved with physical stimulation, airway manipulation (usually jaw thrust), and delivery of high-flow oxygen by facemask.

In our study, 5 (3.7%) patients experienced apnea lasting more than 30 seconds, 3 of whom became hypoxic. One patient was intubated. Four patients recovered, either spontaneously or after physical stimulation, and none of these required BVM-assisted ventilation. Swanson et al reported that 2 (10%) of 20 adult patients experienced transient apnea, 1 requiring BVM ventilation [10]. In the study by Miner et al, 2 (4%) patients in the propofol arm required BVM for apnea, whereas in the study by Coll-Vinent et al [8], 2 (22%) of 9 adults receiving a single 1.5 mg/kg bolus of propofol required BVM for apnea. Clearly, respiratory depression requiring immediate intervention is the predominant complication of ED PS with propofol.

Emesis rarely complicates the use of propofol, probably because of its antiemetic property. In our study, 1 patient experienced emesis with no evidence of aspiration. This patient was intubated. In prior ED studies, there were no reported episodes of emesis [3-10].

Ours is the only ED study of propofol for PS in which a subject required intubation. The patient was an 18-year-old man with a history of mild asthma who underwent PS for reduction of a distal radius fracture. During PS, he developed prolonged apnea, hypoxemia (nadir SpO₂ 75%) and emesis. Bag-valve-mask ventilation was attempted, succinyl choline was administered, and the patient was orally intubated. The patient was extubated in 32 minutes without evidence of adverse sequelae. The development of prolonged apnea at the time of PS was likely related to medications that were given before PS. In the 95 minutes before PS, he received 8 mg of morphine IV and 2 mg of lorazepam IV, as well as immediate premedication with 0.8 mg/kg of fentanyl. The total dose of fentanyl and propofol used and the rate of propofol infusion were similar to the study mean. Refer to Table 1 for details.

An iatrogenic narcotic overdose likely occurred, but lorazepam may have also contributed. In one study that examined the synergistic effects of propofol and benzodiazepines, the dose of propofol required to produce anesthesia was reduced by 52% in the presence of midazolam [18]. Laryngospasm is another possible explanation for this
patient’s course. Laryngospasm is a known complication of propofol that occurs with a frequency of less than 1% [12]. This is a consideration because the provider managing the patient’s airway noted that BVM ventilation was very difficult despite an oral airway being in place and the absence of wheezes. The ventilation difficulty resolved after administration of a paralytic. A third, less likely explanation for this patient’s course is fentanyl-associated rigid chest syndrome.

We chose to administer propofol from a handheld syringe. The initial propofol bolus averaged 0.98 mg/kg. This was followed by intermittent microboluses of 10 to 20 mg based on estimated patient weight, every 30 to 60 seconds, titrated to level of sedation. The mean microbolus infusion rate was 0.22 mg/kg/min (about 14 mg every minute in a 70-kg adult). For procedures lasting greater than 2 to 3 minutes, additional microboluses were usually required when purposeful movement resumed. While explicit dosing guidelines were recommended, dosing parameters were not actually controlled. Consequently, we found that microbolus infusion rates varied substantially, with a range of 0.02 to 0.8 mg/kg/min (from about 1.5 to 55 mg/min for a 70-kg adult).

In contrast to our dosing protocol, early ED studies of propofol for PS delivered propofol by continuous IV infusion, using a pump [3,10]. The slow continuous infusion technique suffers from a longer feedback loop between perceived level of sedation and adjustment of infusion rate. Thus, it is prone to produce undersedation or oversedation. A nurse usually operates the IV pump. The intermittent microbolus technique likely results in tighter control of the level of sedation and requires less nursing involvement. Its main disadvantage is that there is no direct limit on the rate of infusion, and overdose may be more likely to occur by accident or if the anesthetist is impatient.

Whereas 6 prior ED studies appear to have used a similar propofol dosing method to ours, in none of these was dosing precisely described or measured. One study which used a higher bolus dose of 1.5 mg/kg found a very high rate of both hypoxemia (44%) and apnea (22%) [8]. In the large pediatric series by Bassett et al [9], a 1 mg/kg bolus was followed by subsequent doses of 0.5 mg/kg infused over 60 seconds, every 1 to 2 minutes. The study in adults by Miner et al [5] used a similar regimen of a 1 mg/kg initial bolus, followed by 0.5 mg/kg every 3 to 5 minutes. These regimens, in which larger subsequent boluses were administered more slowly and less frequently than in our “microbolus” protocol, produced similar rates of hypoxia and apnea to those in our study.

In our study, physician and patient satisfaction was assessed using descriptive ratings. Please refer to Table 3 for details. We chose not to use a visual analog scale because no comparison was being made. Descriptive ratings were felt to be more meaningful in this setting. In the 129 cases where satisfaction was recorded, physicians rated satisfaction with propofol as ‘excellent’ in 105 (83.2%; 95% CI 74.7%-88.1%). In the 24 cases where physician satisfaction was rated less than excellent, dissatisfaction was noted to be caused by 1 or more of the following: difficulty achieving adequate sedation, excess patient movement, and occurrence of apnea or hypoxemia. Physicians stated they would use propofol again in all but one case.

Patient satisfaction was similarly excellent. Eighty nine percent (95% CI 83.3%-94.3%) of patients did not remember the procedure, and 98.3% stated that they would be willing to receive propofol again. However, 15% (95% CI 8.5-21.2%) of patients rated pain during the procedure as moderate or severe. This finding underscores the need to provide adequate doses of an analgesic agent when using propofol for PS.

Although our study was not explicitly designed to do so, we compared medication dosage between cases associated with adverse events and uncomplicated cases. We found that the propofol bolus dose was significantly higher in the adverse-event group and that there was a trend toward higher propofol infusion rate and initial fentanyl bolus dose. This association between adverse events, such as apnea, and higher propofol and fentanyl dosage is very plausible and likely to be true. Furthermore, close analysis of the single case requiring intubation suggests that benzodiazepines and narcotics may potentiate the respiratory depression from propofol.

Our study suffers from a number of shortcomings. The most significant problems were a wide variation in PS conditions throughout the study and failure to obtain certain data that are considered standard in sedation studies. In an effort to promote the use of propofol by many providers under routine conditions, we purposefully designed a loose study protocol and a brief data collection sheet that focused strictly on dosing, complications, and satisfaction. We did not use research assistants. The trade-off was that we did not closely control study conditions or provider behavior and were unable to gather detailed information on each case. We did not quantify recovery time, although the very short recovery time after sedation with propofol has been firmly established in other studies [8,9]. American Society of Anesthesiology class was not formally assessed and recorded, although patients with abnormal hemodynamics or active cardiopulmonary problems were not considered for PS with propofol. We did not quantify the level of training or prior experience with propofol of the physician providing PS.

In the process of conducting this study, we realized that there were shortcomings in our propofol PS guidelines. Foremost was the lack of required end-tidal CO2 monitoring. Continuous capnography during PS is rapidly becoming standard of care because it is known to detect respiratory depression earlier than physical examination or pulse oximetry [6]. Its routine use during this study might have reduced the incidence of hypoxemia. Second, our PS guidelines should have prompted providers to carefully consider other medications that the patient may have received before PS. In patients receiving benzodiazepines
or narcotics before PS, the propofol dose should be reduced or the procedure delayed.

In conclusion, propofol was effective for deep PS in the ED, when used in a routine fashion for a variety of indications. Physician and patient satisfaction with this form of PS was excellent. However, there was a significant incidence of hypotension, hypoxemia, and apnea, and 1 of 136 patients required intubation. Adverse events were associated with a higher initial bolus dose of propofol. Careful patient selection and scrupulous monitoring during PS with propofol are mandatory.

References

Correspondence

After the black box warning: dramatic changes in ED use of droperidol

To the Editor,

Several recent articles and letters, both in AJEM and elsewhere, have commented on the propriety of the FDA black box warning on the use of droperidol [1-4]. As many emergency physicians believe that the drug’s dangers have been overstated, we sought to determine whether actual ED use has been affected. We distributed a 1-page survey to 260 attendees at the plenary session of a national Emergency Medicine meeting 10 months after the FDA warning was issued. Respondents answered questions regarding their use of droperidol and how the warning changed their practice. Responses were analyzed with Pearson’s chi-square.

A total of 207 surveys were returned, for an overall response rate of 80%. The majority of respondents, 69%, had more than 5 years experience; 82% worked in EDs with a census greater than 25K, and 42% in sites greater than 50K. Fully 97% of respondents were aware of the black box warning and 81% had changed their use of droperidol. Changes in droperidol use were unaffected by practice volume and years of experience. The drug had been remarkably popular; 73% had been using droperidol at least once a week, with 53% using it almost every shift. As a result of the warning, 71% of respondents now never use droperidol. Changes in droperidol use were unaffected by practice volume and years of experience. The drug had been remarkably popular; 73% had been using droperidol at least once a week, with 53% using it almost every shift. As a result of the warning, 71% of respondents now never use droperidol. Only 11% of those discontinuing droperidol cited patient safety concerns; monitoring requirements and hospital formulary discontinuation were both noted by 42%. For those still using droperidol, two thirds have not decreased the dose and only 12% now obtain a pretreatment 12-lead electrocardiogram; cardiac monitoring is used by 14% for varying periods. More than half (57%) of those changing their practice noted using ondansetron as an alternative for nausea/vomiting, perhaps adding credence to the concerns voiced by Mullins and Van Zwieten [1]. Of those using alternative sedative agents (haloperidol, lorazepam, or ketamine), only 8% thought the alternative agent was more effective.

The changes in ED practice caused by the FDA-imposed black box warning [5] represent an excellent example of how clinical practice may be rapidly altered by regulatory action. Our findings demonstrate a dramatic change in ED practice largely unrelated to clinical concern. Indeed, the vast majority of our respondents (92%) believed that their chosen alternatives were no more effective than droperidol; almost half (49%) thought the alternatives were worse. Our survey clearly indicates that the black box warning on droperidol has dramatically decreased its use in the ED, often to agents perceived to be less effective.

References

[5] Inapsine (droperidol injection), black box warning, Taylor Pharmaceuticals, and Akorn Co, 11/01.

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Ultrasound for transcutaneous pacing: documentation, usage, and definition

To the Editor,

I read with interest the article by Holger et al [1] on the use of bedside ultrasound to determine capture in transcutaneous ventricular pacing (TCP). I agree with the authors that when using ultrasound to determine TCP capture, electrocardiogram (ECG) monitoring on the ultrasound machine and video recording are unnecessary. However, without these two options there will be a problem of how to document the capture properly for quality assurance purpose. I wish to suggest the following method as the solution. First, use a 3.5-MHz curvilinear transducer with a small footprint to visualize the heart and the pericardium in B mode using the subxiphoid or parasternal long-axis views to detect pericardial effusion or tamponade. A visual assessment of the overall cardiac contractility is made at the same time. Next, place the electrodes on the patient and begin TCP under real-time ultrasound monitoring. Electrical capture is confirmed in the conventional manner by the appearance of a consistent ST segment and T wave after every pacer spike on the cardiac monitor. Then change the scan setting to B/M mode to display both the 2-dimensional image and the M mode tracing, freeze the image, and calculate the heart rate on the M mode tracing with the machine’s built-in measurement function (Fig. 1). If the measured heart rate corresponds to the set pacer rate then mechanical capture is confirmed. This method allows documentation of the mechanical capture in a single static printout. It does not require any video recording and/or additional set of ECG input to the ultrasound machine.

I agree with the statement in an earlier article by Holger et al that M mode is not superior to 2-dimensional ultrasound for determining capture in TCP [2]. However, in centers where the ultrasound machines do not have ECG input and/or video recording capability, M mode does offer an effective alternate means of determining and

Fig. 1 Documentation of mechanical capture using the B/M mode and built-in heart rate measurement function. IVS indicates interventricular septum; LV, left ventricle; MV, mitral valve; and RV, right ventricle.

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doi:10.1016/j.ajem.2004.01.007
documenting mechanical capture in TCP to fulfill the ultrasound quality-assurance requirement.

In our center, ultrasound has been used successfully for primary confirmation of ventricular capture in TCP in a 70-year-old patient with sinus bradycardia (33 beats/min) and cardiogenic shock 9 days after coronary bypass grafting [3]. In our patient, clinical determination of mechanical capture in TCP was extremely difficult because of the weak pulse volume and the presence of skeletal muscle contractions. Bedside ultrasound enabled us to see the ventricular contractions and heart valve motions corresponding to the pacer rate in real time despite these difficulties. Skeletal muscle contractions did not interfere with obtaining a good ultrasound image during TCP. Although Holger et al did not study the difficulty of making clinical interpretation of ventricular capture or whether clinically indeterminate capture occurred, our case clearly illustrated that the usefulness of ultrasound for confirming mechanical capture in TCP lies exactly in those clinically indeterminate cases.

I would also like to point out that although it may be necessary to define ventricular capture as “observed ventricular wall motion synchronous with the pacing-spike image on the ultrasound screen” for the purpose of study design, it may not be possible to use the same definition in the practice setting for the following reasons. First, determination of electrical capture requires the visualization of ST segment and T wave after every pacer spike, but not all ultrasound machines are equipped with ECG input with dampening circuitry. If the ultrasound machine cannot display paced ECG complexes in its usual form, a separate cardiac monitor and leads have to be used, making the whole setup very cumbersome. Second, many ultrasound machines in use do not have the option of ECG input at all. In these situations, visualization of ventricular contractions with a rate corresponding to the set pacer rate and synchronous with either audible signals from the cardiac monitor or pacer shocks (as evident from thoracic muscle contractions) should suffice as proof of mechanical capture.

References


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Transient ischemic attack associated with Metabolife 356 use

To the Editor,

We present a case of a young, otherwise healthy woman who experienced symptoms of a transient ischemic attack after ingestion of Metabolife 356 (Metabolife International Inc, San Diego, Calif), a supplement marketed for weight loss. Metabolife 356 contains ma huang (stated 12 mg ephedrine), guarana extract (stated 40 mg caffeine), chromium picolinate, and various herbal and vitamin supplements per tablet. Ephedra alkaloids are sympathomimetics, which have been associated with vasoconstriction. In theory, the ephedrine-induced vasoconstriction may have led to this patient’s transient ischemic symptoms.

A 20-year-old woman presented to the emergency department complaining of numbness to her left face, arm, and leg that began 1 hour before arrival. She had a mild headache and nausea. She denied any other similar episodes or prior medical problems. She admitted to ingestion of 4 tablets of Metabolife 356 less than 30 minutes before the episode. She also stated that she ingested 6 to 15 tablets daily for the 3 days prior in an attempt to lose weight. Family history, social history, and review of systems were otherwise negative. Physical examination revealed a well-developed woman in no distress. Heart rate was 89 beats per minute, with a blood pressure of 134/84 mm Hg, respiratory rate of 16 per minute, and oral temperature of 99.1°F. Pupils were 4 mm, equal, and reactive. Neck, pulmonary, cardiovascular, and abdominal examinations were normal. Cranial nerves (II-XII) were intact. Motor was 5/5 in all extremities. Two-point discrimination was decreased throughout her left side. Reflexes were equal and normal bilaterally. Romberg sign and cerebellar signs were normal. Cranial computerized tomography was normal. The patient was admitted and a neurological consultation was obtained.

Electrocardiogram, complete blood count, coagulation studies (prothrombin time/full thromboplastin time, fibrinogen), cardiopulmonary immunoglobulin (Ig) A, IgG, IgM, lupus anticoagulant, antinuclear antibody, and homocysteine levels were normal. Urine EMITTM drug screen for amphetamine, barbiturates, cocaine, ethanol, opiates, propoxyphene, tricyclics, and cannabinoids was negative. Lumbar puncture revealed normal cell counts and culture. Brain magnetic resonance imaging was normal. The patient’s symptoms resolved within 4 hours and she was discharged the following day. No rechallenge was performed.

Ephedrine is an alkaloid derived from the plant genus Ephedra, primarily Ephedra sinica, also known as ma huang. Its medicinal use dates back to ancient China for the treatment of asthma, although introduction into the United States has occurred within only the last century. The past decade, however, has witnessed an explosion in the unmonitored use of ephedrine for not only the treatment of asthma (proportionally very few), but also for weight loss and athletic performance enhancement. As a herbal product or “dietary supplement,” however, regulation and testing of Ephedra is not held to the same strict standards as pharmaceuticals, leaving open a Pandora’s box of potential lack of efficacy, inappropriate dosing, side effects, and adverse events, including death [1,2].

The Ephedra alkaloids act as sympathomimetics, directly and indirectly (through the release of norepinephrine) resulting in stimulation of both α and β receptors. The use of ephedrine in the treatment of asthma is likely via bronchodilation by activation of the β2 receptors. However, the nonselective nature of this stimulant can also result in hypertension, palpitations, nausea, urinary retention, mydriasis, tachycardia, tremors, central nervous system stimulation, and hyperthermia. Adverse event reporting has included cases of psychosis, dysrhythmias, seizure, premature delivery, fetal demise, and cardiac arrest. Ephedrine has also been implicated in a number of ischemic events, and although the specific mechanism is unclear, a combination of hypertension, vasospasm, vasoconstriction, and platelet activation has been purported, resulting in ischemic and even hemorrhagic stroke [1-4].

Ephedra species contain a mixture of alkaloids, including but not limited to ephedrine, pseudoephedrine (commonly used in over-the-counter nasal preparations), and phenylpropanolamine (taken off the market after being linked to hemorrhagic stroke). Its structure differs from that of cocaine and methamphetamine by only a single substitution. In addition, ephedrine is often mar-
keted in combination with caffeine, a methylxanthine that also acts as a sympathomimetic. However, the relative concentrations of either substance are not necessarily standardized between formulations or even between lots of the same formulation.

Of note, as of November 18, 2003, Metabolife International Inc has removed all Ephedra-containing products, including Metabolife 356, from the market, pending further regulatory clarification. Other previously widely available Ephedra products are curiously absent from drug, health food, and grocery stores, although many remain easily obtainable via the Internet. Adverse effect reporting has clearly contributed to the course of ephedrine sales, but as regulation and enforcement continue, perhaps measures to determine safety and efficacy can be taken prospectively rather than using multiple adverse outcomes to establish a pattern of harm.

Limitations of this case report, as in most, include the lack of a rechallenge, which would be unethical in this case.

In conclusion, the use of Metabolife 356 was temporally related to the development of a transient ischemic attack in our otherwise healthy 20-year-old patient.

References

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Can nurses appropriately interpret the Ottawa Ankle Rule?

To the Editor,

With great interest, we have read the article written by Fiesseler et al [1] addressing the topic of interobserver agreement and accuracy of triage nurses concerning the interpretation of the Ottawa Ankle Rules (OAR). Clinical research of this kind is of obvious importance to the development of exploring cost-effective and safe alternatives for conventional treatment in the emergency department (ED). However, in this letter we would like to discuss the applied methodology in relation to the conclusions drawn by the authors.

Although the inclusion criteria were not explicitly mentioned in the article, some concern was raised about the age of trauma included in the study. As stated in the “Methodology” section, no patients were included having sustained injuries more than 1 week old. However, as shown in the systematic review of Bachmann et al [2] earlier this year, assessment of injuries more than 48 hours old significantly influences the sensitivity of the OAR. Therefore, although both observers were confronted with the same injury, it might be better to refrain from assessing injuries more than 48 hours old by the OAR.

One of the conclusions drawn in the discussion of the article is that the poor to moderate $\kappa$ values suggest a low accuracy for the nurses in interpreting the OAR. However, $\kappa$ values represent the agreement between two observers of which in this study, EPs nor triage nurses can be considered the gold standard. In determining the diagnostic accuracy of the observers, the final outcome of OAR assessment should in our opinion be the radiographic proof of presence or absence of fracture (gold standard).

In the “Results” section of the article, positive and negative predictive values for the EPs and nurses are mentioned. To enable extrapolation of these parameters to use them in daily practice, a representative random sample of ankle injuries presented in the ED is necessary. The authors mention that no coherent explanation can be found for the prevalence in their study, they opt that a possible explanation would be that patients of higher socioeconomic status contacted their private physician to bypass long wait periods in the ED. We would like to emphasize that the mentioned positive predictive values are prone to overestimation and the negative predictive values to underestimation because of the high prevalence of fractures in this study and therefore should be interpreted cautiously.

In conclusion, the observational trial by Fiesseler and colleagues presented the interesting finding that interobserver agreement for OAR between triage nurses and EPs is moderate. However, we do not agree with the conclusion drawn by the authors that the moderate agreement results are proof of low diagnostic accuracy for triage nurses. Consequently, the advice to let triage nurses use these rules only under supervision of a physician is in our opinion not supported by the presented results.

References

Correspondence

The Authors’ reply,

We appreciate the interesting and insightful comments of Dr. Derksen regarding our article. Based on the findings of Bachman et al [1], we agree that inclusion of patients within 2 days from injury would have been preferential. However, our data was collected in 2001, 2 years before Bachman’s publication. Also, when Steill et al [2] developed the Ottawa Ankle Rule (OAR), inclusion criteria were limited to those patients presenting earlier than 10 days from injury. We implemented a slightly “more stringent” criteria of 7 days.

With regard to our poor to moderate $k$ values suggesting a lower accuracy for nurses, our conclusions were not based solely on interobserver agreements ($k$ values) but in conjunction with sensitivity overall for missed fractures. Previous studies have validated physician implementation of the OAR [3-5]. We used physicians as a surrogate marker for analysis purposes of the OAR individual criteria. The utility of this was to help determine areas of inconsistency when compared to physicians. Our concern regarding individual $k$ values should only heighten one’s awareness regarding its appropriate and safe implementation by nurses. If nurses are not localizing similar areas anatomically as physicians, these decision rules become less effective.

In response to the utilization of positive predictive values and negative predictive values, we agree that they should be used cautiously in the setting of such a high prevalence of disease. As you previously stated, this can overestimate or underestimate results, respectfully.

The data were not analyzed using fixed- or random-effect models in our study. Analysis using these methods in the setting of multiple observers (ie, many different physicians and nurses) would render our results less meaningful. Pooling of physicians and nurses into groups of each, respectfully, was thought to be the best way to represent our data.

Our conclusion that the nurses should only use these rules to determine the need for radiographs with appropriate supervision is not solely based on $k$ values, but also pertains to the sensitivity rate of 92%. Steil et al [2] stated in the development of the OAR their objective was a sensitivity of 100% for significant fracture. In our opinion the medical legal implications of missing a fracture are not acceptable by physician extenders if utilizing a decision rule not yet validated for this group. Interestingly, the physician’s sensitivity was similar to nurses. This study was not meant to analyze physicians and such, these results should be used cautiously. We feel that our previously stated conclusions are accurate based on the information obtained from our research.

References

Correspondence

Inducing high levels of carbon monoxide in a tent

To the Editor,

We were concerned to read the article by Thomassen and colleagues describing the intentional exposure of healthy subjects to carbon monoxide (CO) inside a closed space [1]. In their study conducted in Norway, 7 men sat inside a tent for 2 hours with a burning kerosene camping stove “to investigate if burning a cooking stove inside a tent is a potential health hazard.”

As background in their article, the authors recognize that CO is formed whenever incomplete combustion of carbon fuels occurs. Furthermore, they describe an earlier report of 2 individuals dying of CO poisoning from using a cookstove in a tent [2]. In the present study, each subject was exposed for 120 minutes although continuous measurement of ambient CO concentrations revealed unsafe levels much earlier.

Emanuel et al have identified 7 requirements that must be satisfied for clinical research to be ethical [3]. This study clearly violates at least one of them, a favorable risk-benefit ratio. This requirement necessitates that risks to the subject are minimized and that they are proportionate to the benefits to the subject and society. At a minimum, the subjects’ exposure in this case could have been discontinued much earlier based on the unsafe levels of CO detected before the planned experimental time had elapsed. Moreover, the subjects’ presence and exposure was unnecessary to demonstrate that the stove would produce CO. The relationship between inhaled CO and carboxyhemoglobin concentration is well known; thus the objective of their study could have been achieved simply by monitoring the levels of CO in the tents [4]. Based on a recent CO poisoning treatment study, these subjects may now have a significant risk for long-term cognitive impairment [5].

Although the authors’ local ethics committee approved the study, it does not meet accepted standards for ethical research. If unethical studies such as this are published, other investigators may be encouraged to pursue similar work and it may cause unnecessary harm to research participants.

References


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Carbon monoxide poisoning from a cooking stove in a tent

To the Editor,

I was interested to read this study [1] that clearly demonstrates dangerous levels of CO within tents—as it is an underrecognized problem with many other reports of poisoning and fatalities [2-12]. The authors are to be congratulated on managing to achieve CO levels of 350 to 500 ppm with one as high as 600 ppm. No previous tent or snow cave study has exceeded 300 ppm [13-16]—the level thought necessary to cause collapse [Ministry of Defense (MOD) unpublished data]—nor has any previous laboratory experiment [5,9,13,17-19] exceeded 300 ppm if the volumes of the experimental models are adjusted to an average tent volume of 5000 L. The high CO levels achieved may paradoxically be because the air channels were left open for ventilation and the zipper was opened at 90 minutes when the stove stalled. During combustion, partial ventilation may actually produce higher CO levels than no ventilation at all [20] by allowing CO production to continue instead of extinguishing the flame, as occurs from lack of oxygen when cooking in an airtight room [18].

The mean COHb of 21.5% is also higher than the 18% achieved by Irving et al [4] in a series of uncontrolled experiments in poorly ventilated tents with a kerosene stove during winter on Mount Washington, New Hampshire, in 1942 and those of 10% found in Pugh’s [5] tent occupants during the 1956 to 1957 Trans Antarctic Expedition. The latter found higher levels of COHb in tent occupants who had been cooking compared to those just burning the stove (10% vs 5%)—as found with CO levels during cooking in this study. Increased CO production during cooking has also been noted by others [5,13,14,16,18-21] and postulated (as did the authors in this study) to be as a result of flame cooling [13,14,18]. However, a recent randomized controlled study failed to show any significant difference (P > .05) in CO production between pans of ice and water when each was brought to boiling point [19]. However, this study did note 3 occasions (each during ice melting) where there was a rapid and significant increase in CO production (P < .014) when the previously controlled flame underwent a yellow flare, and this may represent the same occurrence as the “spike” on the graph that the authors describe when their stove stalled. The same experimental model in a further randomized controlled study found a significant (P = .002) increase in CO production when the pan diameter was increased from 165 to 220 mm [17]. This leads to the hypothesis that it is flame dispersal during cooking—not flame cooling—that leads to increase in CO production as a result of an increase in the absolute level of combustion. This hypothesis is further supported by other observations that CO production also rises when an object such as a pan, rock [20], or aluminium block [21] disperses the flame.

The risks of CO poisoning at altitude are high because of the direct additive hypoxic effect of the COHb [15,22,23], the hyperventilation of altitude [24], the increased endogenous CO production [25], the greater CO sink due to the polycythemia [25], and the lengthened CO half-life [26]. The symptoms can easily be wrongly assumed to be because of acute mountain sickness and the camper should be extra vigilant.

To avoid CO poisoning within tents while cooking, the camper should bear in mind the following recommendations: avoid prolonged simmering, keep the stove highly pressurized and burning with a maximum blue flame, use small diameter pans, be very wary of yellow flares, and if this occurs, turn the stove off, repressurize then reignite and allow maximum tent ventilation for a few minutes. Ventilation should provide an air/oxygen ingress port low down in the tent with an egress port for combustion gases high in the tent to allow laminar ventilatory flow. The camper should be wary of the paradoxical risks associated with inadequate ventilation and appreciate that regular good tent ventilation will be better than continuous inadequate ventilation. If these measures fail and CO poisoning starts to occur, the onset of headache and tachycardia can develop insidiously in the sedentary tent individual. Regular trips outside the tent, which can be used to clear the tent fly sheet of snow/ice buildup, may unmask these symptoms. Let us hope that no other campers succumb to this avoidable killer.

References


0735-6757/$ – see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.ajem.2004.06.012
[23] EPA U, Agency EP. Altitude as a factor in air pollution.
Correspondence

The Authors’ reply,

We appreciate the responses to our article regarding carbon monoxide (CO) poisoning in tents from the use of small cooking stoves [1]. Both comments contribute to increasing the awareness of this underrecognized problem. Dr Hampson and colleague are concerned about our experimental design and judge it to be unethical. Their major concern seems to be that the subjects who participated in the study might suffer a significant risk for long-term effects in the form of cognitive impairment.

This critique is a bit harsh considering that the experimental design mimicked a normal situation occurring under outdoor activities in winter. As experienced mountaineers, all of the subjects also have been exposed previously to similar conditions for several hundred days. Therefore, the experimental exposure did not add significantly to their total exposure. In addition, the main goal for the study was not merely to document that the stoves produced CO. The relation between inspired CO concentrations and carboxyhemoglobin (COHb) is known [2], but we wanted to see if the tents' airflow combined with the subjects’ performing duties normally done in tents. However, we did not fixate the gas sensor close to the nose/ mouth region of the subjects, or combined this with a CO filter mask, which would have been a preferred experimental design solution. The finding of significant amounts of COHb in the blood astonished us based on our previous experience in which we found no symptoms of CO intoxication under similar conditions.

However, we acknowledge the point made that the experiment could have been stopped earlier, when the ambient air measurements of CO exceeded some value. In retrospect, looking at the graphical display of the results, we agree that the experiment could have been terminated after 60 minutes without missing the main findings. On the other hand, as revealed after the analysis of the blood samples, it took more than 45 minutes before the venous COHb levels exceeded 10%.

It is postulated that exposure to high concentrations of CO for a short period is less harmful than exposure at a lower concentration for a longer period [3]. As can be read from our article, none of the subjects experienced any symptoms of CO poisoning, for example, dizziness, light-headedness, or headache 2 hours after the exposure. Dr Hampson and colleague have themselves nicely showed that headache is a sign of CO poisoning, although this is nonspecific [4]. Our study subjects were also extensively informed that they were free to leave the tents at any time if they felt uncomfortable in any way, but none did.

The occurrence of cognitive impairment in CO-intoxicated patients with only headache or nausea as the presenting symptoms seems to be low. Delayed neurological deterioration after CO exposure is also reported to be more rare in younger subjects [5]. Mathieu and colleagues found that only 1 of 96 patients had any manifestations 1 year after the CO poisoning [3]. Weaver et al also studied patients with symptomatic acute CO poisoning [6]. In the period after the experiment was performed, we have had personal contact with all the subjects, of whom none have complained of any symptoms. All of them will also receive a copy of the comments in this issue of the journal. On this basis, we feel that the ethical objections raised by Dr Hampson and colleague are exaggerated.

We are grateful for the large number of interesting references not listed in the Medline database, provided by Dr Leigh-Smith. In the 1942 study on Mt Washington, Irving et al reported that in a “plastic-treated tent,” subjects with a COHb level of 16% felt no significant symptoms [7]. When it comes to the possible reasons for the high levels of CO in our tents, it may be that polyester tents are less permeable than cotton, combined with the high effects of the stoves (approximately 3000 W) and relatively low tent volumes (outer tent made of polyester, approximately 4600 L; inner tent made of polyester cotton, approximately 2700 L). As Dr Leigh-Smith nicely states in the end of his comment, there are many measures that can easily be used by campers to avoid being unknowingly a victim of CO from cooking stoves in tents. Still, there are many unresolved questions regarding how to choose cooking stoves for use in small tents under winter conditions.

References


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Hemoptysis caused by Hughes-Stovin syndrome

To the Editor,

Hemoptysis is a common complaint that emergency physicians encounter. Most cases are minor or self-limited; however, massive hemoptysis occasionally occurs and can be life-threatening. Patients with Hughes-Stovin syndrome can present with massive hemoptysis from pulmonary artery aneurysms and pulmonary embolisms, and specific treatments are required for this potentially lethal condition. Here we report a unique case and discuss the clinical implications for emergency physicians.

A 34-year-old man, with previously healthy status, presented to the emergency department (ED) with hemoptysis for 1 week. The amount of hemoptysis increased gradually in 1 week’s duration despite antitussive treatment. He denied taking any drug that might predispose to bleeding. Physical examinations were unremarkable except superficial venous engorgement over his abdomen and legs. No ulcer was found in his oral cavity or genital region. His platelet count and coagulation tests were within normal limits. A chest radiography revealed rounded opacities in bilateral lung fields (Fig. 1). A subsequent computed tomography (CT) of chest showed pulmonary artery aneurysms in the right lower and left lower lobes with mural thrombi (Fig. 2). Inferior vena cava thromboses were also found in this CT study. A pulmonary ventilation/perfusion scan was performed, which showed bilateral and well-defined ventilation/perfusion mismatched areas, suggestive of a high probability of pulmonary embolism. However, anticoagulants were not given for the pulmonary embolism because of his concomitant hemoptysis condition. Finally, a rare diagnosis of systemic vasculitis, Hughes-Stovin syndrome, was made on the basis of multiple pulmonary artery aneurysms and inferior vena cava thromboses in such a young patient. A magnetic resonance angiography (MRA) was performed to better understand the morphology of pulmonary artery aneurysms, and it disclosed 5 aneurysms of the pulmonary arteries at the right upper, right middle, right lower, left lingual, and left lower lobes, respectively (Fig. 3). A consultation with a cardiothoracic surgeon was obtained and lung transplantation was suggested for the multifocal aneurysms. The hemoptysis resolved with intravenous methylprednisolone treatment. The patient has been well, without a recurrence of hemoptysis, for 1 year.

Hughes and Stovin first described a syndrome consisting of multiple pulmonary aneurysms and peripheral venous thrombosis in 1959 [1]. Since that time, several cases of the Hughes-Stovin syndrome have been published [2-5]. Hughes-Stovin syndrome might be, in fact, a cardiovascular manifestation of Behcet disease or the incomplete type of Behcet disease (isolated pulmonary presentation of Behcet disease) [6,7]. Arterial involvement in Behcet disease was expressed by aneurysms that can involve all arterial territories with a preference for the pulmonary arteries (Hughes-Stovin syndrome) [8,9]. As for the venous involvement, recurrent phlebitis commonly involves the large vessels and results in thrombus formation. Like our case, he presented with engorged abdominal superficial veins, suggestive of vena cava thrombosis. Most patients with Hughes-Stovin syndrome have unstable pulmonary artery aneurysms, which may result in massive hemoptysis. However, some patients have concomitant pulmonary embolisms within the aneurysms, probably because of an inflammatory response of the vascular endothelial cells [8-10].

Imaging studies for diagnosis of pulmonary aneurysm are straightforward, such as a chest radiograph or a CT [11]. For better evaluation of the aneurysms in Hughes-Stovin syndrome, conventional pulmonary angiography remains the best tool. However, it cannot be performed in patients with thromboses in the inferior vena cava, because the thromboses impede catheter passage. In these cases, contrast-enhanced 3-dimensional MRA may be a substitute for visualizing the aneurysms, as in our case [12].

Once the Hughes-Stovin syndrome is recognized in the ED, a prompt treatment with immunosuppressant, either systemic corticosteroids or cytotoxic agents, confers clinical improvement and stabilizing of the pulmonary artery aneurysms in most patients [5,6,13]. However, serious hemoptysis occasionally recurs despite these treatments. Moreover, if the pulmonary embolism is present in the aneurysms, this condition may pose a therapeutic dilemma in using anticoagulants. Anticoagulation prevents the progression of pulmonary embolism, yet it increases the risk of hemoptysis. A study of isolated pulmonary Behcet disease presenting with hemoptysis showed that 4 patients treated with anticoagulants for pulmonary embolism con-
continued to have hemoptysis [13]. One of them subsequently died after massive hemoptysis, despite good anticoagulant control. Based on these observations, we think that using anticoagulants should be reserved only for patients with embolisms in the main pulmonary artery that causes hemodynamic instability.

If the pulmonary artery aneurysms rupture in the ED and life-threatening massive hemoptysis follows, it may necessitate mechanical ventilatory support [5]. A consultation with cardiothoracic surgeon should be obtained immediately. The affected segments of the lung should be resected if applicable, predicated on the preoperative MRA or angiographic findings [12]. If the aneurysms are identified in the bronchial arteries, bleeding can be treated by embolization of bronchial arteries [4,14].

In summary, patients with Hughes-Stovin syndrome can present to the ED with unstable pulmonary aneurysms and hemoptysis. Although massive hemoptysis is infrequent, it carries a very serious prognosis. This report underscores the potentially lethal condition of Hughes-Stovin syndrome for emergency physicians. Appropriate treatments with immunosuppressant and aggressive surgical intervention are imperative for the life-threatening hemoptysis.

References


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Use of emergency ultrasound in a rural ED with limited radiology services

To the Editor,

Disparity in healthcare delivered to patients seen in emergency departments (EDs) comes in many forms. Studies have suggested differences in care based on ethnicity and education [1]. Inequities in healthcare occur in other specialties as well, such as the discrepancies in rates of revascularization attempts in women and men with myocardial infarction [2]. Medical care may also differ based on geography. For example, access to mammography for women living in remote areas differs significantly from that available to women in suburban areas [3]. For emergency physicians (EPs) working in large tertiary referral centers, few barriers to advanced imaging care may exist. However, this is not the case for many rural hospitals [4].

With the development of emergency ultrasound (EU), EPs practicing in rural settings have a viable, partial alternative to patient transfer or admission because of limited radiologic services. As the number of EPs trained in EU increases, the rural practitioner can become more diagnostically self-sufficient. Our study objective was to determine whether introduction of EP-performed ultrasound (US) affects patient management and disposition in a rural ED when faced with limited radiology services.

This was a prospective observational study over 4 months surveying EPs on effects EU has on decision making in a rural ED with limited radiology services. Sampling was based on study physician availability at this 7000 annual census ED. The study was approved by the Institutional Review Board. Radiology reading was available one morning per week. The hospital had plain radiography and computed tomography (CT) but no teleradiology. A Medison US machine with curved linear and linear array transducers with power and spectral Doppler was available. Emergency ultrasound hospital credentialing was based on the 2001 ACEP guidelines [5].

Two EPs were asked to fill out surveys regarding patients requiring an US before the scan. The EPs recorded the reason for examination, availability of alternative imaging, radiologist availability for interpretation, if transfer would otherwise be necessary, and prescan differential diagnosis. After the US, the EPs recorded the results, differential diagnosis postscan, disposition, and if patient management was altered by EU. Statistical analysis included descriptive statistics and paired t test with 95% confidence intervals (CIs), using StatsDirect (StatsDirect Software, Aswell, UK).

Forth-three EUs were performed (Table 1). Mean patient age was 38 (range, 12-81) with 42% female. The mean differential diagnosis before the US was 2.7. After EU, the mean number for the differential diagnosis fell to 1.2. The decrease of 1.5 (95% CI 1.15-1.73) was statistically significant, \( P < .0001 \). In 44% of cases, alternate imaging was possible, but in only 1 case was a radiologist available. Mean transfer distance was 49.5 miles. In 9% of cases, final EU diagnosis was not part of the original differential diagnosis. This included identification of a testicular mass on US for suspected testicular torsion, intra-abdominal fluid in suspected renal colic, gallstones on US for an AAA, and discovery of incarcerated femoral hernia while ruling out a leg deep vein thrombosis (DVT). Management was changed in 74% (32) of cases. In 53% (25) of cases transfer was avoided.

Almost half of all acute care hospitals in the United States are considered rural. Rural populations have a higher prevalence of acute and chronic illness, higher level of poverty, higher level of unemployment, and lack of insurance compared with their urban counterparts [6]. These factors lead to fewer services at rural hospitals such as high-cost technical equipment as well as personnel trained in its use [7]. Lack of radiology services in rural settings results in transfer of patients to urban/larger hospitals for diagnostic testing [4].

Various methods have been proposed to provide a higher level of service to rural populations. Teleradiology, the transmission of diagnostic images between 2 institutions, is becoming more common [8,9]. However, this technology does not obviate interpretation delays and can be very expensive. In emergent cases such as AAA or ruptured ectopic pregnancy, teleradiology may be impractical as rapid diagnosis is essential [10-12]. If EPs are to affect the outcome in these patients, delay in diagnosis is unacceptable. Further, in contrast to CT, US is highly operator-dependent and thus is less suited for teleradiology.

The scans performed during this study clarified the clinical situation by decreasing the differential diagnoses in most cases. Furthermore, in 9% of cases, EU yielded
diagnoses not originally part of the differential diagnosis, and patient management was changed in 74% of cases. In cases where transfer was avoided, not only did the patient potentially benefit by avoiding an unnecessary transfer but the local community as well. Only 1 ambulance was available in the county at once and a transfer removed the only crew from this community for 2 or more hours. Only 2 EPs performed EUs and the patient population was small. There was no correlation with other diagnostic studies. However, the physicians involved are credentialed for EU and routinely perform EU examinations without confirmatory studies. The range of pathology scanned may vary by setting. In summary, EPs using US in a rural ED with limited radiology frequently changed management and avoided transfer.

References


### Table 1

<table>
<thead>
<tr>
<th>Patient Type</th>
<th>Study Type</th>
<th>Disposition</th>
<th>Effect on Management</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>LE</td>
<td>Admit</td>
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</tr>
<tr>
<td>2</td>
<td>FAST</td>
<td>Transfer</td>
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</tr>
<tr>
<td>3</td>
<td>FAST</td>
<td>Observe</td>
<td>Avoided transfer for CT</td>
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<td>4</td>
<td>Ocular</td>
<td>Discharge</td>
<td>Avoided transfer for evaluation</td>
</tr>
<tr>
<td>5</td>
<td>Renal</td>
<td>Discharge</td>
<td>Avoided transfer for CT</td>
</tr>
<tr>
<td>6</td>
<td>LE</td>
<td>Transfer</td>
<td>Led to transfer for IVC filter</td>
</tr>
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<td>7</td>
<td>GB</td>
<td>Admit</td>
<td>Avoided transfer</td>
</tr>
<tr>
<td>8</td>
<td>Testicular</td>
<td>Transfer to OR</td>
<td>Avoided transfer led to transfer for operative intervention</td>
</tr>
<tr>
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<td>Vascular</td>
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<td>Avoided admission for observation</td>
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<tr>
<td>15</td>
<td>Pelvic</td>
<td>Transfer</td>
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</tr>
<tr>
<td>16</td>
<td>LE</td>
<td>Transfer</td>
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</tr>
<tr>
<td>17</td>
<td>LE</td>
<td>Admit</td>
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</tr>
<tr>
<td>18</td>
<td>Bladder</td>
<td>Discharge</td>
<td>Avoided Foley placement</td>
</tr>
<tr>
<td>19</td>
<td>Testicular</td>
<td>Discharge</td>
<td>Avoided transfer for US evaluation</td>
</tr>
<tr>
<td>20</td>
<td>AAA</td>
<td>Admit</td>
<td>Avoided transfer for CT r/o AAA</td>
</tr>
<tr>
<td>21</td>
<td>FAST</td>
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<td>Avoided transfer for evaluation</td>
</tr>
<tr>
<td>22</td>
<td>AAA</td>
<td>Transfer</td>
<td>Changed transfer from air to ground</td>
</tr>
<tr>
<td>23</td>
<td>Pelvic</td>
<td>Discharge</td>
<td>Avoided transfer for r/o ectopic evaluation</td>
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<tr>
<td>24</td>
<td>Testicular</td>
<td>Transfer</td>
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<td>26</td>
<td>GB</td>
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<tr>
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<td>28</td>
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<td>Avoided transfer for US r/o DVT or heparin</td>
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<td>Avoided transfer for US DVT evaluation</td>
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### Table 1 (continued)

<table>
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<th>Effect on Management</th>
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<tr>
<td>37</td>
<td>AAA</td>
<td>Transfer</td>
<td>Changed planned air transport to ground</td>
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<td>38</td>
<td>GB</td>
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<td>Avoided transfer for GB US evaluation</td>
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<td>GB</td>
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<td>Avoided admission for antibiotics and observation</td>
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<td>Transfer</td>
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<td>Avoided transfer for CT r/o of AAA</td>
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<tr>
<td>42</td>
<td>AAA</td>
<td>Discharge</td>
<td>Avoided transfer for CT r/o of AAA</td>
</tr>
<tr>
<td>43</td>
<td>Pelvic</td>
<td>Transfer</td>
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</table>

Abbreviations: LE, lower extremity; FAST, focused assessment with sonography in trauma; GB, gallbladder; AAA, abdominal aortic aneurysm; Echo, echocardiogram; IVC, inferior vena cava; DVT, deep vein thrombosis; CT, computed tomography.


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Missed diagnosis of acute exertional compartment syndrome, occurring after a short run

To the Editor:

A 19-year-old previously fit infantry soldier developed pain on the anterior aspect of his right leg during a 3-mile run. The run was conducted in appropriate training shoes, and he was familiar with this distance. After the first half mile, he developed pain in his shin. He continued for the next half mile and had to stop due to the pain. His medical officer, concerned that the pain was out of proportion to the history, referred him to the local accident and ED. On questioning, he did not report previous such pains. No fall or awkward landings took place during the run, and the pain had developed gradually. On examination, the right calf and shin were tender, and dorsiflexion was painful. Passive extension of the compartment was not tested. Radiographs were taken showing no bony injury or signs of stress fracture. The casualty officer diagnosed a muscle sprain, and he was sent back to barracks on crutches with analgesia.

Over the next 2 days, the pain increased while he rested in his bed. His medical officer was again concerned and referred him directly to a local surgical unit. At this stage, he had a tense tender anterior compartment with overlying redness and purpuric skin changes. He was unable to dorsiflex the ankle. Passive extension was painful, and sensation was altered over the dorsum of the first web space.

Fasciotomies of the compartments of the right leg were performed. The contents of the anterior compartment were obviously necrotic, and debridement was carried out. Further debridement was carried out at 48 hours resulting in complete excision of tibialis anterior and more than 90% of the remaining muscle mass in the compartment. Inspection and closure followed after a further 48 hours. He underwent extensive rehabilitation; however, he was left with a permanent foot drop and sensory changes.

Compartment syndromes occur when the volume of the content of a muscle compartment is increased resulting in interstitial pressure rising above the capillary perfusion pressure. A cycle of ischemia, edema, and further rises in pressure continues. Acute compartment syndromes occurring after exercise are rare; however, they have been described in various forms [1,2]. Exertional compartment syndromes are proposed to be caused by acute muscular hypertrophy and edema with microhemorrhages in the exercised muscles [3]. Most often, the condition has been described where the intensity or duration of the exercise is greater than previously experienced such as soldiers pushing themselves to meet fitness test standards [4], but it has been reported in a professional sportsman [5]. Often, it may be preceded by a history of chronic episodes of pain rapidly resolving after cessation of the exercise. In our case, the soldier involved was well used to this type and level of exercise and had experienced no previous anterior leg pains.

A high level of suspicion is required to make the diagnosis in this unusual presentation when any leg pain has no definite diagnosis. The symptoms described are out of proportion to what might be expected from the history. The pain may come on anything from a few minutes into the exercise up to developing 12 hours after. It often increases gradually to a constant deep intense pain unrelieved by analgesia. Passive stretching of the compartment muscles elicits a painful response. The compartment may be visibly tense and swollen. Measuring intracompartmental pressure is useful in confirming the diagnosis. Normal levels range from 0 to 15 mm Hg, but there is no consensus on when fasciotomy is required. Pressures exceeding 30 to 35 mm Hg have become accepted as levels indicating fasciotomy; however, levels should be assessed along with the patient’s systemic blood pressure. Levels within 40 mm Hg of the mean arterial pressure [6] or within 20 mm Hg of diastolic pressure [7] have been proposed.

Having said this, the clinical symptoms and signs remain paramount to making the diagnosis.

Acute exertional compartment syndrome is rare, and without a high level of suspicion, the diagnosis can be missed with disastrous consequences. This case of a previously fit soldier suffering pain in his leg a short distance into a run illustrates this point. Even with knowledge of this condition, the diagnosis was difficult to make because of the unexpected short distance of the run provoking symptoms. Without another firm diagnosis, this syndrome should be suspected and managed with complete compartmental surgical decompression. The use of intracompartmental pressure measurement could be a useful adjunct to diagnosis.
References


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A pregnant woman presenting to the ED with Valentino’s syndrome

To the Editor,

Pregnant patients with acute abdominal pain can pose a diagnostic and therapeutic puzzle for the emergency department (ED) physician. The general reluctance to use conventional radiographic study and the modification of both symptoms and signs because of anatomic and physiologic alterations caused by pregnancy may delay or mislead the diagnosis. Peptic ulcer in pregnancy is rare, and acute perforation is even more rare. Perforated peptic ulcer in pregnancy can be miserable to both mother and fetus if not promptly recognized and timely remedied. The following is a case with Valentino’s syndrome—unusual presentation of perforated peptic ulcer at ED.

A 23-year-old woman in the 20th week of gestation visited our ED with the complaint of abdominal pain in the right lower quadrant (RLQ) for 3 days. She had a history of duodenal ulcer without any treatment. The pain was initially in the epigastric area for 1 day and then shifted to the RLQ in the following 3 days. The character of the pain was persistent but not cramping. There was no fever, diarrhea, and constipation. She denied any other systemic diseases, major operations, daily medication, or history of allergy. On physical examination, vital signs revealed only a heart rate of 119 beats per minute. The abdomen was soft, but there was tenderness and rebound tenderness in the RLQ area and epigastric area. The degree of tenderness in the RLQ was more severe than in the epigastric area. The obturator sign was present, but psoas and Rovsing’s sign were absent. The bowel sounds were hypoactive.

Laboratory data revealed a white blood cell count of 11.9 $\times$ 10$^3$/μL with 77% neutrophils and 5% bands, hemoglobin level of 11.5 g/dL, and platelet count of 154 $\times$ 10$^3$/μL. Emergency ultrasonography revealed small amounts of fluid accumulation around the cecum without obvious tubular structure and abnormal gynecologic finding. With acute appendicitis suspected clinically by the surgeon, she underwent appendectomy via the McBurney incision. During the operation, turbid ascites approximately 50 mL was aspirated, but unexpectedly, the appendix was grossly normal. The surgeon performed a laparotomy, and a 0.3 $\times$ 0.3 centimeter sealed-off perforated duodenal ulcer on the anterior wall of the bulb was detected. The appendix revealed periappendicitis in the pathological report. The postoperative course of the mother and fetus was uneventful. She was discharged from the hospital 12 days after admission.

Gastrointestinal surgical problems occur in approximately 0.5% to 1% of all pregnancies [1]. Peptic ulcer disease is uncommon in pregnancy. A reduction in gastric acid secretion and gut motility combined with increased mucus production has been proposed as the explanation [2,3]. Acute complications of peptic ulcer, such as perforation, in pregnancy are more rarely encountered. The manifestation of perforated peptic ulcer is sudden, sharp, and severe abdominal pain. At first, it is located in the epigastrium but quickly spreads over the entire abdomen, especially along the right side of the abdomen, as the chemical peritonitis. When the duodenal contents descend to the right pericolic gutter, it can mimic appendicitis (Valentino’s syndrome) [4]. Physical examination may reveal rebound tenderness and guarding of the abdomen. Laboratory study findings reveal leukocytosis. An erect abdominal radiograph may demonstrate gas under the diaphragm [5]. Because of the hesitancy of radiographic studies during pregnancy, and the low incidence of the disease and similar symptoms in healthy pregnant women, the diagnosis of perforated peptic ulcer can be difficult. The mortality rate in these patients is high, and the fatal outcome appears because of late diagnosis and operation. Acute appendicitis is the leading cause of the acute abdomen in pregnancy, occurring in approximately 1 in 1000 to 1500 pregnancies [6,7]. Appendicitis typically presents as epigastric or periumbilical pain and the pain migrated to RLQ, the single most reliable symptom [6]. The accuracy of clinically preoperative diagnosis of appendicitis in pregnancy ranges from approximately 60% to 80% [8,9]. The ultrasonography is a good tool to assist us to diagnose acute appendicitis in pregnant women at ED. If the appendix appears normal during operation, it is crucial to search for other causes [5,6].

According to the presentations, imaging studies in our patient and disease prevalence, we reasonably suspected acute appendicitis. However, actually these presentations were also Valentino’s syndrome in pregnancy. On reviewing the literature, this was nearly never been reported in

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doi:10.1016/j.ajem.2004.04.037
pregnant women before. In summary, if a pregnant patient with peptic ulcer history suffers from acute RLQ pain, ultrasonography demonstrates pericecal fluid accumulation without obvious blindended tubular structure, despite rarity, Valentino’s syndrome should be taken into consideration.

References

Correspondence

Adrenal hemorrhage mimicking an acute abdomen

To the Editor,

A 72-year-old man with a history of colonic diverticulitis, jejunal diverticulosis, and bronchoemphysema complained of right flank pain, 39°C fever, and asthenia. He was lethargic and hypotensive, and his abdomen was diffusely tender with bowel noises absent. White blood count was 12900, and C-reactive protein was 156 mg/L (<10). Activated prothrombin time was markedly increased at 100 seconds (25-40 seconds). Septic shock of abdominal origin was suspected, leading to a diagnostic laparoscopy that was noncontributive. Postoperatively, the patient continued to be severely hypotensive despite the administration of large amounts of colloids and saline. Subsequent onset of hyperkalemia led us to suspect acute adrenal failure which was confirmed by the presence of bilaterally enlarged adrenal glands on computed tomography scan (Fig. 1) and hyperintense nodules on magnetic resonance T1-weighted and T2-weighted sequences (Fig. 2). Baseline plasma cortisol (15.6 mmol/L [165-790]) and aldosterone (<2.5 ng/L [10-310]) levels were low. Antiphospholipid syndrome as a contributing factor was established by a positive screening test for IgG anticardiolipin. The patient made a spectacular recovery within a few hours after initiation of steroid replacement therapy.

Bilateral adrenal hemorrhage is a rare cause of acute adrenal failure. Medical or surgical stress and therapeutic anticoagulation are the usual triggering factors. In a significant proportion of patients, a circulating lupus anticoagulant is also present [1-3]. The nonspecific clinical manifestations associated with this condition are difficult to recognize especially in a postoperative context. Symptoms include abdominal, flank or back pain, fever, hypotension, and lethargy [1,3]. Adrenal hemorrhage can mimic an acute abdomen [2]. Blood analysis may show hyperkalemia, hyponatremia, azotemia, acidosis, leukocytosis, or a sudden decrease in hematocrit [1]. The diagnosis is confirmed by computed tomography scan or magnetic resonance imaging revealing bilaterally enlarged and spontaneously hyperdense/hyperintense adrenals, features that are indicative of recent hemorrhage [1,2]. Biochemical confirmation can be obtained by measuring baseline cortisol and corticotropin levels or by a corticotropin stimulation test, but this should not delay initiation of steroid replacement therapy. Typically, clinical improvement is spectacular within several hours.
References


Correspondence

Vibrio vulnificus—a rare but fulminant pathogen causing airway obstruction

To the Editor,

Vibrio vulnificus is one of the most invasive and rapidly fatal human pathogens known and was first associated with disease in human beings in 1970 [1]. Two clinical syndromes were reported: the first is virulent primary septicemia, occurring in persons who have eaten raw seafood; the second form is a severe, rapidly progressive wound infection in patients who have had contact with seawater or who have sustained a wound while handling or cleaning shellfish. A life-threatening illness may develop in susceptible individuals, particularly in patients with preexisting liver diseases [2]. V. vulnificus–caused upper airway obstruction was not reported before. We report a 60-year-old man with alcoholic liver cirrhosis and diabetes developed parapharyngeal (PP) and retropharyngeal (RP) abscesses with septic shock caused by V. vulnificus 10 hours after exposure to raw seafood.

A 60-year-old man presented to the ED at 6 AM with fever, dyspnea, and bilateral neck swelling. He had no history of penetrating wound in his neck or oropharynx. One day before coming to ED, he ate raw swordfish and some undercooked shrimp at about 7 to 8 PM for dinner. Medical history was notable for alcoholic liver cirrhosis and type II diabetes.

Physical examination revealed sick-looking and mildly jaundiced man with bilateral neck swelling. His temperature was 38.2 °C, respiratory rate 20 breaths per minute, pulse rate 105 beats per minute, and blood pressure 70/50 mm Hg. Initial laboratory findings were: white blood cell count 5700/mm³ (with a differential of 78% segmented forms and 7% band forms), serum bilirubin was 7.5 mg/dL (with direct bilirubin of 4.6 mg/dL), aspartate aminotransferase 212 U/dL, alanine aminotransferase 85 U/dL, and C-reactive protein 172.6 mg/dL. Chest radiographs were normal. Flexible laryngoscope showed swollen epiglottis, bulging lateral pharyngeal walls, and a narrow airway. After intravenous fluid challenges, nasoendotracheal tube was inserted under flexible bronchoscopic guidance. Contrast-enhanced head and neck computed tomography (CEHNCT) showed abscesses in bilateral PP and RP spaces (Fig. 1A, B).

Emergent cricopharyngomyotomy was done through cervical incisions in the middle of the neck bilaterally to open the PP and RP spaces. At operation, the subcutaneous tissue and muscle appeared grossly edematous and oozed serous fluid. There was no evidence of skin or muscle necrosis. Teichoplanin (6 mg/kg/d), cefazidime (120 mg/kg/d), and metronidazole (32 mg/kg/d) were given intravenously after operation.

Blood cultures obtained at ED and pus culture obtained at operation grew V. vulnificus which is sensitive to ampicillin, amikacin, aztreonam, cefazidime, ciprofloxacin, ceftriaxone, cefuroxime, gentamicin, imipenem, piperacillin, and sulfamethoxazole-trimethoprim. The antibiotics were shifted to cefuroxime (45 mg/kg/d) and gentamicin sulfate (3 mg/kg/d) for 3 days, and cefuroxime was used alone for 7 more days. The patient’s condition improved gradually, and he was discharged.

V. vulnificus is a common organism found in tropical marine environment [3]. Raw and uncooked oysters or shellfish are vehicles for infection of V. vulnificus [3]. Chang et al [4] described that the most common symptoms in primary septicemia caused by V. vulnificus were fever (100%), chills (93%), and hypotension (79%). In our patient, the initial manifestations as airway obstruction and the stormy course with sepsis syndrome are unique. It is believed that ingestion of seafood served as a vehicle for the entry of the pathogen with a subsequent extraintestinal spread causing PP and RP abscesses.

In our patient, the duration between intake of seafood and appearance of shock was about 10 hours. The development of the disease was rapid. Blake et al [2] described a V. vulnificus septicemia illness that had begun 24 hours after ingestion of raw oysters. V. vulnificus sepsis is associated with a mortality rate greater than 50%, and it may exceed 90% in patients with shock within 48 hours of admission [5]. The possible mechanism behind these could be abundant endotoxins and exotoxins from V. vulnificus which induce cytolysis of the host’s cells and septic shock by stimulating the production of tumor necrosis factor–α [6]. Aggressive medical and surgical treatment could hinder the progression of the disease.

CEHNCT enables diagnosis and accurate localization of PP and RP spaces abscesses. In V. vulnificus caused PP and RP abscesses, treatment should include maintenance of airway, hemodynamic stability, proper antibiotics, and prompt surgical intervention. Surgery includes emergent drainage and debridement of infectious foci. In selection of
antibiotics, Chuang et al [7] advocated that third-generation cephalosporin should be the drug of choice for early antimicrobial therapy. Ceftazidime plus doxycycline, cefotaxime, or fluoroquinolone was also recommended by other authors [8]. In this patient, ceftazidime, teichoplanin, and metronidazole were used initially for broadest spectrum coverage and were proved to be effective.

In summary, *V. vulnificus* should always be suspected as a pathogen, even in rare locations, in liver cirrhosis patients who had contacted raw seafood recently. Detailed history taking and aggressive medical and surgical treatment could lower the mortality rates.

**References**


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Atrial septal defect: an important risk factor after trauma

To the Editor,

Asymptomatic patients with atrial septal defects (ASD) may be at increased risk of serious complications after trauma when right-to-left shunt and severe hypoxia occur acutely because of increased pulmonary artery pressure.

A 21-year-old man, presented fully conscious with a fractured femur after a deceleration injury while driving at 60 mph. No clinical injuries or radiological abnormalities of the neck, chest, or abdomen were identified. Electrocardiogram showed sinus rhythm. Hematologic and biochemical tests were normal.

On day 2, an intramedullary nail in the right femur was inserted.

On day 3, the patient’s condition deteriorated. He was pale, with temperature 38.5°C, blood pressure 85/45 mm Hg, and heart rate 140 beats per minute, and his jugular venous pressure was raised. Chest examination revealed a decrease in breath sounds at both lung bases and diminished heart sounds and pulse oximetry (SpO₂) was 94% (FiO₂ 0.4). Arterial blood gas showed H⁺ 46 mmol · L⁻¹, PaCO₂ 5.5 kPa, PaO₂ 10.7 kPa, bicarbonate 21.7 mmol · L⁻¹, and base excess −3.9 mmol · L⁻¹. Chest x-ray showed a markedly enlarged heart (Fig. 1), and electrocardiogram showed low-voltage complexes with T-wave inversion in the chest leads and a new right bundle-branch block. Platelet count was 68 × 10⁹ · L⁻¹, hemoglobin 7.2 g · dL⁻¹, and international normalized ratio 2.8. Fresh frozen plasma, platelets, and blood were transfused. Biochemistry tests showed urea 6.2 mmol · L⁻¹, creatinine 192 μmol · L⁻¹, K⁺ 5.9 mmol · L⁻¹, creatinine kinase 2546 IU · L⁻¹, markedly elevated alanine transaminase 2246 IU · L⁻¹(normal 5-40 IU · L⁻¹), alkaline phosphatase 94 IU · L⁻¹(normal 40-110 IU · L⁻¹), and total bilirubin 54 μmol · L⁻¹(normal <17 μmol · L⁻¹). Meanwhile, oliguria ensued and later anuria. No myoglobinuria was detected.

Transesophageal echocardiography (TOE) was performed, and 1.5- to 2-cm ASD was confirmed. Troponin I 8 ng · mL⁻¹(normal <0.5) was reported.

During days 4 and 5, inotropes requirement decreased, but FiO₂ remained high (0.8). Large bilateral pleural effusions were identified, and chest drains were inserted on both sides.

An ultrasound of the abdomen showed an enlarged liver, but liver enzymes and bilirubin level decreased (alanine transferase 295 IU · L⁻¹, alkaline phosphatase 90 IU · L⁻¹, and bilirubin 32 μmol · L⁻¹).

On days 6 and 7, FiO₂ decreased (0.4), and inotropes were weaned. TOE showed a significant improvement in right ventricular function, with a huge right atrium. The ASD was smaller in size. Renal function was improving, and continuous venovenous hemofiltration was discontinued.

During days 9 and 10, ventilatory support was weaned, and the trachea was extubated.

Transesophageal echocardiography reported an elevated right ventricular systolic pressure (50 mm Hg). Treatment with ramipril (angiotensin-converting enzyme inhibitor) was started.

At 2 months’ cardiology clinic review, the patient was asymptomatic, and a transthoracic echocardiogram showed reasonable function of both ventricles and an ASD (smaller in size) was identified with left-to-right flow (no shunt reversal).

The sequence of events pointed to the possibility of fat embolism and cardiac contusion.

Cardiac tamponade is an important differential diagnosis after trauma and deceleration injuries particularly with a raised troponin I; however, elevated troponin I is not exclusive of myocardial contusion, and cardiac contusion is not necessarily related to the degree of thoracic trauma [1]. TOE offered an excellent diagnostic test [2] to exclude cardiac tamponade.

Major pulmonary embolus was excluded by computed tomography scan of the chest and pulmonary angiogram.

Central and peripheral cyanosis ensued, and further deterioration in renal function and acidosis occurred. Tracheal intubation and ventilation became necessary. Continuous venovenous hemofiltration and hemodynamic support with inotropes and nebulized prostacyclin were started.

During days 9 and 10, ventilatory support was weaned, and the trachea was extubated.

Transesophageal echocardiography (TOE) was performed, and 1.5- to 2-cm ASD was confirmed. Troponin I 8 ng · mL⁻¹(normal <0.5) was reported.

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The patient fulfilled most of the major and minor criteria, as identified by Gurd and Wilson [3] for the diagnosis of fat embolism syndrome. Petechial rash was not seen, but retinal hemorrhages were identified. Although pathognomonic when present, petechial rash is not essential to diagnose fat embolism [4].

Compromised right ventricular function and pulmonary artery dilatation were shown by echocardiography [5], but major pulmonary embolus was excluded.

The detection of ASD by echocardiography and TOE enlightened the pathological sequence of events highlighting the possibility of paradoxical embolisms.

Foramen ovale is anatomically open in 25% of individuals but functionally closed by the higher pressure in the left atrium. Right-to-left shunt and subsequent paradoxical embolism may occur when pressure in the right atrium rises [6]. Complicated prognosis could result when larger emboli reach the systemic circulation [7].

In our patient, an acute right-to-left shunt was a result of the increase in the pulmonary pressure in the right side of the heart most likely as a consequence of fat embolism.

The acute deterioration of liver function can be explained as a consequence of heart failure and an increased systemic back pressure.

Cyanosis in this case could indicate Eisenmenger syndrome, but it could also be caused by unusual direction of tricuspid regurgitant flow (as shown by echocardiography) increasing the right-to-left shunt [8].

In conclusion, the presence of chronic ASD is an important risk factor after trauma that could precipitate complicated events. A high index of suspicion is essential to achieve a successful outcome.

References

Correspondence

Syphilis screening in a high-risk, inner-city adolescent population

To the Editor,

During the syphilis epidemic of the 1990s, several authors advocated that high-risk adults should undergo screening for syphilis in the emergency department (ED) but did not comment on adolescents [1,2]. Adolescents, however, are more likely to engage in high-risk activities such as unprotected sex and to have multiple sexual partners. The Centers for Disease Control and Prevention stated that, “The primary behavior leading to a high risk of syphilis transmission is unprotected sex of an individual or a group of individuals with multiple partners” [4]. For adolescents, the ED provides relative anonymity and easy access for complaints related to a sexually transmitted disease (STD); the ED is therefore an expected point of presentation [1].

Between 1996 and 1998, Baltimore, Md, had the highest rate of primary and secondary syphilis in the United States [5]. In 1999, 3 cities and 22 counties accounted for half of all cases of primary and secondary syphilis in the nation [3,6,7], and Baltimore was among the top 3 cities [7]. In 2000, half of all primary and secondary cases of syphilis were concentrated in only 21 counties and the city of Baltimore [8].

A retrospective review was performed in an inner-city tertiary care hospital. Patients enrolled in the study were adolescents who presented to the pediatric ED between April 1999 and April 2001 with clinical evidence of STD (as defined in Table 1). At the time of the study, standard ED practice for adolescents undergoing evaluation for STDs was to screen with a rapid plasma reagin (RPR) test. Adolescents with positive qualitative RPR test results underwent confirmation with a quantitative RPR test and a fluorescent treponemal antibody (FTA) test. Patients excluded from the study were those younger than 12 years or older than 18 years and those evaluated for sexual assaults.

Between April 1999 and April 2001, a total of 902 adolescents met the inclusion criteria (the age distribution is shown in Table 2, and 574 (64%) were screened for syphilis with an RPR test. A total of 6 of the 574 follow-up logs indicated positive RPR test results: 2 patients had positive FTA results (one incident case and one case of known syphilis), 3 had negative FTA results (false-positives), and 1 was lost to follow-up. The incidence of newly detected syphilis was 0.17% (1 of 574) and the prevalence was 0.35% (2 of 574) (Table 3). The incident case was in a 17-year-old who had a titer of 1:8 (with the quantitative RPR test) and weakly reactive FTA test results.

At the University of Maryland, the cost of a qualitative RPR test is US$13.87. Patients with a positive qualitative RPR result undergo quantitative RPR testing, at a cost of US$17.35, and an FTA test, at a cost of US$29.51. In our study, one incident case of syphilis was detected at a laboratory charge of US$8219.03.

In the present study, the charge for the laboratory tests alone was US$8219 per detected case (this incident case had an RPR titer of 1:8 and a weakly positive FTA test result). This figure does not include the ancillary ED costs of screening for syphilis. By comparison, prior studies have determined the cost of screening to be between US$104.90 and US$251 per case detected [1,2,9].

Table 1 Criteria used for screening for STD

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower abdominal pain</td>
<td>Evidence of epididymitis</td>
<td></td>
</tr>
<tr>
<td>Vaginal discharge</td>
<td>Penile discharge</td>
<td></td>
</tr>
<tr>
<td>Genital lesions</td>
<td>Genital lesions</td>
<td></td>
</tr>
<tr>
<td>Other clinical evidence of STD</td>
<td>Other clinical evidence of STD</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Age distribution of the 902 adolescents who met the inclusion criteria of the study

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>13</td>
<td>24</td>
</tr>
<tr>
<td>14</td>
<td>50</td>
</tr>
<tr>
<td>15</td>
<td>102</td>
</tr>
<tr>
<td>16</td>
<td>147</td>
</tr>
<tr>
<td>17</td>
<td>232</td>
</tr>
<tr>
<td>18</td>
<td>334</td>
</tr>
</tbody>
</table>
The limitations of our study include the following: first, the sampled population was in an urban area with one of the highest rates of syphilis in the country, but the incidence of syphilis decreased during the study period. From 1990 to 1999, the rates of primary and secondary syphilis in the nation decreased by 88%, which may have resulted in fewer cases detected. Second, although the rate of other STDs in the 328 patients who did not undergo RPR testing is similar to that of the 574 patients who had an RPR test, the incidence of syphilis in the 328 patients is unknown. Third, if the single incident case of syphilis had not been discovered, it is unknown how many additional cases of syphilis would have been generated or what the cost of those cases would have been.

These data do not support a recommendation to screen for syphilis in all adolescents undergoing evaluation for STDs in a pediatric ED. However, further studies may elucidate more specific risk factors that identify subgroups of adolescents who should undergo screening for syphilis in the ED.

Acknowledgment

We thank JA Dietrich, MD, JW Lassiter, MD, JA Roberson, and JR Hanna, MD, for help and support with data gathering.

References


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Outpatient delayed screening for patients with suspected deep vein thrombosis

To the Editor,

Venous thromboembolism is estimated to affect approximately 2 million Americans per year and is a frequently encountered problem in the emergency department (ED) [1]. Although venography is the “gold standard” test for diagnosing deep vein thrombosis (DVT), it has been mostly replaced by noninvasive testing using ultrasound (US). In many areas of the United States and Canada, there is a shortage of technicians trained to perform these studies. As a result of this shortage, technicians will quit if they are repeatedly called in at night. This has caused some vascular laboratories to refuse doing scans at night. Because many patients present after normal business hours when no technician is available, emergency physicians are faced with either admitting all patients with suspected DVT, holding them all night in the ED, or allowing selected patients to be followed up as outpatients. It has been previously shown that patients with DVT can be safely treated as outpatients using low-molecular-weight heparins [2-4]. Bauld and Kovacs [5] proposed using low-molecular-weight heparin to avoid admission before US investigation in patients with suspected DVT.

Starting in 1999, our hospital instituted a clinical guideline of delayed investigation of patients with suspected DVT who present after normal business hours. Patients were risk stratified using the scale proposed by Anand et al [1]. If the emergency physician felt that a patient was at high risk for DVT or complications, the patient was admitted for heparinization and screened with US in the morning. If a patient was not at high risk and immediate US was unavailable, the patient was given an injection of enoxaparin (1 mg/kg to a maximum of 150 mg/kg) and discharged to return in the morning for a US of the affected extremity. Before being given an injection of enoxaparin, a patient is supposed to have a platelet count measured. A low platelet count was a contraindication for the use of enoxaparin. Ultrasound was available from 9 AM to 5 PM, 7 days a week including weekends and holidays.

During 2001, a total of 101 patients had delayed outpatient screening US using this guideline. Of the 101 patients, 5 had DVT and 3 had superficial thrombophlebitis. There were no problems with bleeding complications identified from the chart review. In the 5 patients in whom DVT was diagnosed, based on a chart review of their admission, there were no pulmonary emboli. Although the protocol was well received by both emergency physicians and vascular US staff, there were problems with protocol violation. Thirty-nine patients did not have a screening complete blood count to exclude a low platelet count. Twenty-one patients did not receive enoxaparin, including one patient who was ultimately diagnosed with DVT. The reason given in the chart for not giving the enoxaparin was very low suspicion of DVT. There were 5 patients who did not have their scan performed within 12 hours as proscribed in the protocol. One of these patients did, in fact, have a DVT and was not scanned for 2 days.

Although it is optimal to be able to screen all patients with suspected DVT during their emergency visit, it simply cannot be done in every institution. The use of this protocol may help alleviate ED overcrowding. This study did reveal problems with guideline compliance that must be addressed and corrected. Unfortunately, this study was limited by its small size, retrospective design, and use of an unproven protocol.

In conclusion, we found that delayed outpatient screening for DVT in selected patients at lower to moderate risk functioned well in a large suburban hospital. We avoided unnecessary admissions and overuse of the US technician after hours. Further large prospective studies are needed to prove the safety of this approach.

Acknowledgment

The authors thank the staff of the Bethesda North Vascular Lab for their support.

Supported by a grant from the Bethesda Foundation of Cincinnati, Ohio.

0735-6757/$ – see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.ajem.2004.03.004
References


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Urachal abscess: a cause of adult abdominal pain that cannot be ignored

To the Editor:

Omphalitis has once been an important cause of illness and death among neonates throughout the world [1,2]. It is an uncommon disease of the adult. It may be presented with abdominal pain and only mild redness of the surrounding of umbilicus. It cannot be ruled out in adult patients who complain of abdominal pain at emergency department (ED). We report a man with a case of urachal abscess, who presented to our ED with abdominal pain for 1 week.

A 52-year-old man was quite well before. He suffered from periumbilical pain for about 1 week. The character of the pain was not cramping but persistent. Because the pain became more severe, he visited our ED for help. There were no fever, diarrhea, vomiting, and constipation in the recent 1 week. He denied any systemic diseases, major operations, daily medication, or allergic history. On physical examination, blood pressure was 120/80 mm Hg, the body temperature was 36.2°C, the heart rate was 76 beats/min, and the respiratory rate was 18 breaths/min. The abdomen was soft, but tenderness and focal rigidity over umbilical area was found. The bowel sound was normoactive. Others were unremarkable.

Laboratory data revealed white blood cell count of 10.48 × 10³/µL, hemoglobin level of 14.0 g/dL, and platelet count of 241 × 10³/µL. Blood biochemistry was normal. The standing abdomen x-ray film was normal. He was referred for emergency abdominal ultrasonography revealing a hypoechoic tract from the umbilicus to abdominal wall (Fig. 1A) and a hypoechoic mass with heteroechogenic content between the peritoneum and the muscle layer (Fig. 1B). The computed tomography (CT) of abdomen arranged later showed focal inflammation with localized abscess formation over the umbilicus, and the surrounding fat planes were infiltrated (Fig. 2). Under the impression of omphalitis with abscess formation, history was traced. According to the patient’s statement, he habitually dug and scratched his umbilicus daily when he took a shower. Discharge from umbilical region was noted 2 to 3 times per year and it usually subsided spontaneously. However, 1 week before admission, he began experiencing abdominal pain. Spontaneous extrusion of the abscess occurred after admission and the pus culture revealed *Burkholderia cepacia*. He was treated with intravenous amoxicillin/clavulanate 1.2 g every 8 hours. The abdominal ultrasonography that followed showed regression of the abscess, and the total resolution occurred on day 26.

Omphalitis, infection of the umbilical cord and/or the surrounding tissues [2], is uncommon in the adult patient and it presented as a slow-paced and milder clinical course than that of newborn [3]. The causes of omphalitis in the adult are not clear, except trauma and abnormalities of the urachus [4]. Greig and Shucksmith [5] excised 6 of 7 cases of omphalitis; they found no sebaceous glands or hair follicles in the umbilical cavity [3]. They found sweat

![Fig. 1](image1.png) A, Longitudinal scanning over the umbilicus demonstrated a hypoechoic tract (arrow). B, Transverse scanning over the umbilicus demonstrated hypoechoic mass with heteroechogenic content between the peritoneum and the muscle layer of abdominal wall.
glands within the umbilical cavity and the sweat ducts were surrounded by a round cell infiltrate [3]. They proposed that it might progress to dermatitis, stenosis at the skin surface, and abscess formation thereafter [3]. Erythema, edema, and tenderness of the tissues surrounding the umbilicus suggest the diagnosis of omphalitis [6] or urachal abscess. Constant periumbilical pain with or without foul-smelling umbilical discharge may be the initial presentation as our case. Associated signs such as fever, lethargy, and appetite change may implicate systemic complications [6]. Urachal lesions are now better imaged by ultrasonography and CT than by any other image modalities. Demonstration of an abscess within the extraperitoneal fat space of abdominal wall and extension to the umbilicus with or without umbilical discharge is a clue to the diagnosis of urachal abscess [7]. The differential diagnosis of urachal abscess should include hematoma, urachal carcinoma, sarcoma of the abdominal wall [8], peritoneal tumor [9], metastatic carcinoma [10], ventral or umbilical hernia, and inflammatory lesions [11]. Omphalitis may be caused by bacteria or fungus [12]. Pus cultures collected by swabs for both aerobic and anaerobic bacteria after proper skin decontamination are recommended. If there are obvious systemic signs, blood cultures should also be taken [6]. Although infection of the cord stump is rare, its potential sequelae such as cellulitis, necrotizing fasciitis, peritonitis, multiple hepatic abscess, septicemia, and possible retroperitoneal abscess may be fatal [6,13]. Simple omphalitis, without evidence of periumbilical spread, responds to local application of antibiotic compresses or ointment [6]. Systemic antibiotics are indicated if the discharge is purulent or if any evidence of periumbilical spread appears [6].

In summary, omphalitis/urachal abscess may only present as abdominal pain without obvious erythematous periumbilical tissue or exudates in adults. In spite of rarity in adult patient, it should not be ignored in differential diagnosis of abdominal pain. History taking and detailed physical examination may aid us to early diagnosis at ED. Ultrasonography is a good noninvasive diagnostic tool for suspected cases of urachal lesions and good for following up the response of the medical treatment.

References

Correspondence

Isolated extrahepatic bile duct rupture in blunt abdominal trauma

To the Editor,

Single lesion of the main bile duct in closed abdominal trauma is an uncommon event [1], usually produced in patients with severe abdominal trauma involving several organs [1-5]. The diagnosis is difficult unless obtained immediately in a laparotomy performed for another indication [1-5]. We present a new case and discuss the injury mechanisms, diagnostic methods, and different therapies that can be applied.

We present the case of a 36-year-old woman with a history of appendectomy, cholecystectomy (1997), residual choledocholithiasis resolved by endoscopic retrograde cholangiopancreatography, and a biliary prosthesis (2000, subsequently withdrawn). She arrived at the hospital after an accidental fall in her bath with a blow to the anterior abdominal wall, especially the right side. She reported abdominal pain and nausea, without vomiting. At the examination, she was conscious, oriented (GCS of 15), and hemodynamically stable (BP, 130/90 mm Hg). The abdomen was soft with no peritonism. Laboratory results were unremarkable except for hemoglobin (16.7 mg/dL). Abdominopelvic computed tomography scan showed free subhepatic liquid in the right abdomen and vesicorectal space, with no injuries to the spleen, liver, or renal parenchyma. At 12 hours, she presented with peritonism and a surgical exploration was carried out, revealing biliary peritonitis secondary to rupture of the anterior surface of the middle third of the extrahepatic bile duct Fig. 1. The bile duct rupture was closed, and a Kehr tube was inserted at 2 cm from the rupture. The postoperative course was satisfactory. The Kehr tube was withdrawn at 20 days after the surgery.

Lesions of the extrahepatic bile duct and/or gallbladder are produced in 1% to 5% of abdominal trauma [1], and 85% of these are penetrating trauma, usually knife or gunshot wounds [1-5]. The gallbladder is injured in 85% of these patients, whereas injury of the main bile duct alone occurs in 15% (ie, 0.3%-0.5% of patients with abdominal trauma). It is more frequent in young males [1]. The parts of the main bile duct that are attached to other abdominal structures are the most susceptible to injury: bifurcation of the hepatic ducts and intrapancreatic bile duct [1-5]. Main bile duct lesions are commonly associated with severe abdominal injuries. The liver is injured in 50% to 80% of cases [1,4].

Bile duct lesions due to blunt trauma are produced by traffic accidents (compression by safety belt or airbag) [4] and also by falls, kicks, or work accidents [1,2]. Their origin is currently considered multifactorial, although always involving a traumatic force that raises the liver and drops the hepatoduodenal ligament [1,2,4]. Other factors implicated include an increasing pressure on the bile duct transmitted to the interior of the main bile duct [1,3], the functional status of the sphincter of Oddi, and the type of traumatic agent [3]. The portal vein and hepatic artery are not usually injured, because they are longer and more elastic than the main bile duct [1]. If the trauma is major enough to section vascular structures, it is usually mortal [1]. In this case, the temporary placement of the prosthesis and

Fig. 1 Black arrow shows an orifice in extrahepatic bile duct.
cholecystectomy probably prolonged local adherences, facilitating the lesion.

There are 3 injury patterns [1,2]: immediate diagnosis in patients undergoing laparotomy for accompanying injuries [1,4], late diagnosis in stable patients with scant symptoms (>50% of cases) [1], and late complications in patients not diagnosed at the time of the trauma [1,4].

The diagnosis is usually made in an emergency laparotomy for associated abdominal injuries, when bile is observed in the abdominal cavity or bleeding and/or hematoma in the portal pedicle [1,4]. If an immediate laparotomy is not performed the diagnosis is often considerably delayed [1-3]. In fact, there has been an increasing proportion of late diagnoses due to the growing adoption of nonsurgical approaches to the initial treatment of abdominal trauma [6]. In these cases, ultrasonography or abdominal computed tomography shows intra-abdominal liquid considered blood and not bile [3]. A worsening or nonimproving clinical situation, as in our case, can lead to a reassessment of the initial diagnosis. If percutaneous drainage or puncture lavage shows the liquid to have elevated concentrations of amylase and bilirubin, the diagnosis of main bile tract injury is confirmed [1,3]. Hydroxy iminodiacetic acid scan, cholangiRM, and endoscopic drainage can be useful in patients who are not immediately diagnosed [1,2].

When the lesion involves at least 50% of the main bile duct circumference, it is treated by choledochorrhaphy and the insertion of a Kehr tube through a different orifice [1-5]. This is a rapid and efficacious technique, given that the patients do not normally present with dilation of the duct. Various types of patch have also been used to close the defect with variable outcomes [1]. Hepaticojejunostomy is the approach of choice if a complete section of the duct is produced [1,3,5]. A highly selective group of patients, hemodynamically stable and with scant symptoms, can be treated with sphincterotomy, insertion of biliary prosthesis, and percutaneous drainage of the existing bilomas.

The morbidity associated with main bile duct lesions is approximately 10% [1], with reports of biliary fistula, hemobilia, biloma, intrahepatic abscesses, stenosis, and cholangitis [1,2,5]. The mortality in these patients is usually produced by other injuries than the biliary lesion.

References


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Health services use by older adults in an urban public health system

To the Editor,

In addition to extensive use of the emergency department (ED) [1,2], older adults consume a disproportionate share of health care services in both ambulatory [3] and hospital settings [4]. We found no prior studies that place older adults’ use of the ED in the context of use of ambulatory care. The purpose of this investigation was to describe the use of the ED by a large cohort of older adults and compare the use of acute care services with the use of primary care services within the same health care system.

This study was a retrospective cohort analysis using data routinely collected and stored by a comprehensive electronic medical record. For each inpatient, ambulatory clinic, and ED encounter at any site within the targeted health care system, the Regenstrief Medical Record System routinely collects and stores clinical data [5].

The study population consisted of all patients aged 65 years and older who visited our system’s ED or primary care clinics (PCCs) in 2001. We divided the study subjects into 3 cohorts. The first consisted of patients seen in the ED in 2001 but not seen in 1 of the 11 PCCs in 2001. The second cohort included patients with at least one visit to one or more of the PCCs in 2001 but no ED visit in 2001. The third cohort included patients with both a primary care and an ED visit in 2001. Primary care included the ambulatory clinics staffed by general internal medicine, family medicine, or medicine-pediatric physicians. We also obtained data regarding the use of all medical specialty and surgical clinics in our system. For comparative purposes, we obtained ED data for young adults, defined as patients aged 18 to 64 years.

This study was conducted within the Wishard Health System, a university-affiliated, urban, public health system that consists of (1) an ED with 106,000 annual visits, (2) a hospital with 260 staffed beds, (3) 11 primary care centers, and (4) multiple outpatient specialty clinics. Wishard is the only tax-supported public health care facility in Indianapolis.

We used \( \chi^2 \) tests to test for differences in dichotomous measures of demographics and health services use across the 3 cohorts, whereas analysis of variance models were used to test for differences of the continuous measures. In addition, we used \( t \) tests to test for differences for several comparisons that were valid for 2 groups. We presented charge data in actual dollars; however, all statistical tests were performed using the log transformation of the charge data. Charge data were transformed by adding 1 to all values and then taking the natural logarithm.

### Table 1  Demographic data, clinical characteristics, and prior health services use of older patients visiting the ED only, the PCC only, or both

<table>
<thead>
<tr>
<th></th>
<th>ED population</th>
<th>PCC population</th>
<th>ED and PCC population</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of unique patients</td>
<td>1488</td>
<td>2925</td>
<td>2243</td>
<td></td>
</tr>
<tr>
<td>Number of ED visits by the above patient</td>
<td>2391</td>
<td>0</td>
<td>5265</td>
<td></td>
</tr>
<tr>
<td>Number of PCC visits by the above patients</td>
<td>0</td>
<td>9028</td>
<td>11,923</td>
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<tr>
<td>Age (range)</td>
<td>74.2 (65-84)</td>
<td>73.0 (65-99)</td>
<td>72.8 (65-83)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Sex (% female)</td>
<td>52.5</td>
<td>69.3</td>
<td>70.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Race (% black)</td>
<td>46.0</td>
<td>57.1</td>
<td>60.7</td>
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</table>

**Health services use—prior year**

<table>
<thead>
<tr>
<th></th>
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<th>PCC population</th>
<th>ED and PCC population</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean number of hospitalizations in the prior year</td>
<td>0.3</td>
<td>0.1</td>
<td>0.4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>% with any hospitalization prior year</td>
<td>12.6</td>
<td>8.6</td>
<td>22.4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean number of unique prescription drugs from any source</td>
<td>2.1</td>
<td>8.1</td>
<td>11.6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean outpatient charges in prior year</td>
<td>$1563</td>
<td>$1952</td>
<td>$4143</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean inpatient charges in prior year</td>
<td>$15613</td>
<td>$7499</td>
<td>$22,609</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

\[ \text{a} \] Patients aged 65 years and older who visited the ED in 2001 but did not have a PCC visit in 2001.

\[ \text{b} \] Patients aged 65 years and older who visited one of the PCCs in 2001 but did not have an ED visit in 2001.

\[ \text{c} \] Patients aged 65 years and older who visited both the ED and one of the PCCs in the year 2001.

\[ \text{d} \] The prior year was defined as the 365 days before the subject’s index visit in the year 2001.
In a single year, there were 3731 different older adults cared for in this ED with a total of 7656 ED visits. More than 40% of elderly ED patients arrived by ambulance as compared to 18% of patients aged 18 to 64 years. The mean time in the ED for older patients was 7 hours 16 minutes with 27% admitted to the hospital, as compared to 4 hours 30 minutes and 7.6% admitted for younger adults.

Table 1 shows the demographic data, clinical characteristics, and prior health services use of the 3 older adult populations. Patients identified as users of both the ED and the PCCs had the greatest health services use in the year before the visit and were prescribed the most medications. Notably, 40% of all older ED patients were not seen in the health system’s PCCs in the prior year, and 33% were not seen in the system’s primary care or specialty care clinics.

Prior studies have identified older adults as disproportionate users of the ED [6]; however, this is the first to put the use of emergency services by geriatric patients in the context of use of primary care services. Access to regular primary care is considered an important indicator of quality health care systems [7]. Our investigation found that many older adults using this urban ED do not receive care in this health care system’s primary or specialty care clinics.

Within our health system, older patients with both ED and PCC visits used the most health services. Similarly, Byrne et al [8] reported that frequent ED users had more frequent visits to their general practitioner in the prior year. Hansagi et al [9] found that the number of clinic visits significantly increased with increasing frequency of ED visits.

In conclusion, the ED serves as a major contributor to the health care of the elderly within our health care system. Additional study is needed to clarify the proportion of older urban, public ED patients who lack primary care access and elucidate the extent to which patients present to the ED because of a lack of primary care.

References


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Nontraumatic tension pneumocephalus—a differential diagnosis of headache at the ED

To the Editor,

Pneumocephalus is a rare disease, referring to the presence of intracranial air. It occurs secondarily to fracture or surgery on the base of the skull, thoracotomy, tumor invasion, or encephalocele [1]. Tension pneumocephalus is the severe form of pneumocephalus in which intracranial air causes mass effect. Early diagnosis and emergent surgical intervention are mandatory to prevent rapid deterioration of the neurological status. We report a case of tension pneumocephalus, presenting only with severe headache at the emergency department (ED).

A 58-year-old man experienced severe headache 4 months before visiting our ED. The pain was dull and persistent in character and was localized in the bilateral frontotemporal area. No fever, nausea, vomiting, or neurological deficits such as weakness, numbness, and blurred vision were noted. The pain could be relieved partially by analgesics. However, rhinorrhea occurred, followed by aggravation of the headache since 2 days ago. So he visited our ED for help. Reviewing his medical history, we found that he has hypertension under medical control for 4 years. He denied trauma history in the past 4 months.

On arrival, his vital signs were as follows: blood pressure 106/65 mm Hg, heart rate 104/min, and body temperature 36.3°C. On physical examination, his neck was rigid. Neurological examination revealed intact cranial nerves, and the muscle power was full and symmetric. Others were unremarkable. Laboratory data revealed leukocytosis (13,560/μL) with 83% neutrophils. Computed tomography of the brain showed bilateral frontotemporal subarachnoid air accumulation, compressing the brain (Fig. 1). Tension pneumocephalus was impressed and burr hole drainage was immediately performed. Postoperation course was uneventful and his headache subsided. Magnetic resonance imaging showed a skull base cleft at the anterior roof of the sphenoid sinus with cerebrospinal fluid (CSF) leakage (Fig. 2). Functional endoscopic sinus surgery for the repair of the defect was done. He was discharged with a stable condition.

Pneumocephalus is defined as the presence of air in the intracranial cavity. In a review of 295 cases of pneumocephalus, 75% were caused by head injury and cranial surgery, and 8.8% were resulted from infection by gas-forming organism, the majority of which occurred in patients with a predisposing factor such as trauma, paranasal sepsis, or otitis media [2]. But rare cases of pneumocephalus secondary to frequent Valsalva’s maneuver, skull bone invasion of nasopharyneal carcinoma, or unknown causes had also been reported [3]. Tension pneumocephalus occurs when the intracranial air causes mass effect and then the brain is compressed. It usually results from craniotomy or craniectomy. An incidence of up to 12% of patients with secondary to spontaneous CSF rhinorrhea has also been reported [4].

Headache and altered consciousness are the most frequent symptoms of pneumocephalus [5]. Subarachnoid air can cause significant irritation; so as little as 2 mL of subarachnoid air has been reported to cause headache [6]. Tension pneumocephalus usually manifests as deterioration of consciousness, restlessness, generalized convulsion, or focal...
neurologic deficit [7], according to the severity of the mass effect and intracranial pressure [8]. Cardiac arrest caused by tension pneumocephalus has been reported in a patient who had undergone posterior fossa surgery in the sitting position [9].

There are 2 possible mechanisms that pneumocephalus develops. One proposes that air passes through the dural tear by a ball valve effect in which air can be forced into the intracranial cavity by a rapid increase in intrasinus pressure that occurs during coughing, sneezing, or straining, and then the air is trapped intracranially. The other proposes that CSF leakage permits air to enter the intracranial cavity because negative pressure is created as CSF fluid leaves the space [10]. In our patient, both mechanisms may result in the development of tension pneumocephalus.

Computed tomography is superior for diagnosis because it can exclude other pathologies and easily detect air collections (as small as 0.5 mL) in the skull [11]. However, further image studies such as magnetic resonance imaging or cisternography might be required for the identification of the etiology of pneumocephalus.

Pneumocephalus may not require surgical intervention because air might be absorbed spontaneously. However, emergent evacuation of air and reduction of pressure for patients with tension pneumocephalus should be performed to prevent neurologic deterioration.

Tension pneumocephalus is a rare entity, but it should be considered in patients with severe headache at the ED, especially those with concomitant rhinorrhea.

References


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To the Editor,

Obturator hernia is a relative rare mechanical disorder and usually difficult to diagnose preoperatively [1-3]. Although some etiologic factors and clinical characteristics relevant to the diagnosis of obturator hernia have been described [4-6], the typical clinical manifestations are rarely encountered. The mortality associated with obturator hernia has been reported as high as 38% and is caused by delay in diagnosis and surgical treatment [1,2]. The rapid and safe way to establish the diagnosis and management of abdominal emergencies is mandatory. We demonstrate the use ultrasonography in detecting occult obturator hernia, which presented as an acute abdomen and intestinal obstruction in the ED.

An 85-year-old woman visited the emergency room because of severe abdominal pain that lasted for 2 hours. She had a similar episode 1 year earlier at which time she was diagnosed with stool impaction. She had a history of chronic obstructive pulmonary disease and no previous operative history. The intermittent pain localized over the periumbilical area but settled in the lower abdomen without radiation. On arrival, the blood pressure was 150/90 mm Hg, body temperature was 36.5°C, and heart rate was regular at 80 beats per minute. Physical examination revealed distended abdomen, hyperactive bowel sounds, and severe tenderness over the lower abdomen. The white blood cell count was 12,500 cells/μL, and serum amylase was within normal limits. Urine analysis was unremarkable. No abnormal signs were found on rectal and vaginal examinations; there were no masses palpable in the groin. The initial radiograph of the abdomen revealed dilated small bowel loops in the lower abdomen (Fig. 1). Emergent ultrasonography examination was performed, and a loop of bowel in the obturator canal was noted (Fig. 2). Ultrasonography diagnosis of obturator hernia with intestinal obstruction was made. The patient underwent emergency surgery; the obturator hernia was confirmed at minilaparotomy. The postoperative course was smooth, and she was discharged and followed up in the outpatient clinic.

Abdominal pain with ileus is a common problem encountered in the ED. Obturator hernia accounts for about 2% of all hernia [1]. The majority of patients are women, with low body weight and in the seventh and eighth decades of life [1,2,5,6,8]. One explanation could be that emaciated women have a wider pelvis and a more oblique obturator canal [6]. If the diagnosis and management are delayed, it will result in a serious untoward outcome. Prompt diagnosis and management of obturator hernia are mandatory in the ED.

For the diagnosis of obturator hernia, the clinical picture of intestinal obstruction, a positive Howship-Romberg sign, a palpable mass in the groin area, and a previous history of attacks are helpful [5,6]. Barium enema, fluoroscopy [7], computerized axial tomography [8,9], and herniography [10] have all been described in the diagnosis of obturator hernia but sometimes may not be easily immediately available. The correct diagnostic rate varies from 0% to 70% in the patients recorded in the literature [3,9]. In most patients, the diagnosis was made only at the time of laparotomy [2,3]. In obturator

Fig. 1  Plain abdominal radiogram reveal dilated bowel loop.
hernia, plain radiography provides no specific findings. The initial plain abdominal film of our patient did reveal some clue for intestinal obstruction, apart from a dilated bowel loop, the bowel air extended beyond the pelvic floor, but this is not specific for obturator hernia and provided little help in determining the cause of ileus.

There is a general agreement that when a obturator hernia is identified, an initial attempt with nonoperative reduction is indicated for patients without signs of peritonitis or gangrene [10]. If noninvasive reduction is unsuccessful, operative treatment should be immediately performed [11-13]. The reported delay in diagnosis and operative mortality for patients with obturator hernia is up to 33% [1,2]. As in our patient, an elderly multipara, ultrasonography proved capable of identifying the occult obturator hernia that caused her intestinal obstruction. The correct preoperative diagnosis allowed limited surgical approach, thereby reducing surgical risk, especially in the elderly, who poorly tolerate prolonged anesthesia and major surgery.

We demonstrated a patient with the initial presentation of abdominal pain with an ileus pattern on the initial plain radiogram. Using ultrasonography, occult obturator hernia can be diagnosed promptly and may compensate for the limitation of physical examination, thereby avoiding use of other invasive diagnostic modalities. It is widely used to evaluate acute abdomen and also serves as a good diagnostic tool for emergency physicians in evaluating occult obturator hernia in the ED.

References


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Major sport championship influence on ED gender census

To the Editor,

Television major sport championships have been shown to decrease ED patient visits, especially if a local team is involved [1]. Specifically, these events decrease overall visits by 18% and low acuity visits by 30% [2]. The effects of these events on the gender distribution of ED visit have never been studied.

A retrospective review of the ED registration log was conducted on the number of adult gender visits to a New York City hospital with separate psychiatric and nonpsychiatric emergency facilities during the 5 broadcast game times of the millennium World Series between the New York Yankees and the New York Mets. We reported the number of male and female visits for each facility as well as totaled these results for an overall gender census. As a comparison, we repeated this process over the next 4 weeks using the same game period and day of the week.

During the televised games, the ED visits totaled 98 with 8 (8%) psychiatric visits and 90 (92%) nonpsychiatric visits. Out of the 98 visits, 63 (64%) were female and 35 (36%) were male. For the 8 psychiatric visits, 7 (87%) were female and 1 (13%) was male. For the 90 nonpsychiatric visits, 56 (62%) were female and 34 (38%) were male.

The ED visits for the next 4 weeks using the same broadcast time and day of the week as the World Series games totaled 361 with 24 (7%) psychiatric visits and 337 (93%) nonpsychiatric visits. Out of the 361 visits, 180 (50%) were female and 181 (50%) were male. For the 24 psychiatric visits, 10 (41%) were female and 14 (59%) were male. For the 337 nonpsychiatric visits, 170 (50%) were female and 167 (50%) were male.

Statistical comparison between the series group and the nonseries group yielded a P value equal to .041 for gender visit differences to the psychiatric ED, a P value equal to .046 for the nonpsychiatric ED, and a P value equal to .011 for the total ED. A summary of these results is shown in Table 1.

Males comprise 53% to 63% of the fans attending a major sport event and 57% to 70% of the television viewers of a major sport event [3,4]. In major league baseball, males make up 54% of the fan attendance and 70% of the television viewers [3]. Similarly, in the National Football League, males comprise 57% of the fan attendance and 70% of the television viewers [3,4]. This gender discrepancy also occurs for professional women sporting events. For example, males comprise 55% of the Women’s National Basketball Association television viewers, 57% of the women soccer television viewers, and 67% of the Ladies Professional Golf Association television viewers [4].

Because there is a large male interest in watching sports, we expected a decrease in male visits to the ED during the broadcast of the millennium World Series games. Our data support this expectation. For the psychiatric ED census, there was a 71% decrease during the series week. For the nonpsychiatric ED census, there was a 19% decrease. For the total ED census, there was a 22% decrease.

Because females tend to have a much lesser interest in watching sports, we expected the games to have no or very minimal impact on their visit numbers. In other words, we expected the weekly number of female visits to the ED to be roughly equal during the series and the nonseries period. Instead, we found an increase in the number of female visits to the ED during the broadcast of these games. For the psychiatric ED census, there was a 180% increase. For the nonpsychiatric ED census, there was 32% increase. For the total ED census, there was a 40% increase. The exact mechanism for this finding of increased visits is unknown. A possible theoretical explanation for this finding is that females may interpret more social stress during this period, thus, leading to increased psychosocial disturbances, which then results in higher medical use [5–9].

Our study shows that this combination of decreased male usage and increased female usage during the series games

Table 1 Emergency department gender census

<table>
<thead>
<tr>
<th></th>
<th>Series group</th>
<th>Nonseries group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychiatric ED</td>
<td>Female visits</td>
<td>7</td>
<td>10 (2.5/wk)</td>
</tr>
<tr>
<td></td>
<td>Male visits</td>
<td>1</td>
<td>14 (3.5/wk)</td>
</tr>
<tr>
<td>Nonpsychiatric ED</td>
<td>Female visits</td>
<td>56</td>
<td>170 (42.5/wk)</td>
</tr>
<tr>
<td></td>
<td>Male visits</td>
<td>34</td>
<td>167 (42/wk)</td>
</tr>
<tr>
<td>Total ED</td>
<td>Female visits</td>
<td>63</td>
<td>180 (45/wk)</td>
</tr>
<tr>
<td></td>
<td>Male visits</td>
<td>35</td>
<td>181 (45/wk)</td>
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Presented at Society Of Academic Emergency Medicine, Abstract number 81, May 2002 in St. Louis, Mo.
shifts the ED census to a female preponderance when compared to the nonseries period. For the psychiatric ED, the female census went from the nonseries 41% to 87%. For the nonpsychiatric ED, the female census went from 50% to 62%. For the overall ED, the female census went from 50% to 64%. Limitations of the study include its retrospective, observational, and single institution design.

In conclusion, the presence of a nearby televised major professional sport championship shifts the ED census to a female preponderance.

References


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