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Original Contributions

Is prehospital blood glucose measurement necessary in suspected cerebrovascular accident patients?

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Received 20 June 2003; revised 7 August 2003; accepted 12 January 2005

Abstract The present study was completed to establish an epidemiologic database defining prehospital management of suspected cerebrovascular accidents (CVAs) with attention to blood glucose measurement, in the hope of developing recommendations for further treatment protocols. On review of 9495 paramedic run reports for the 24-month period from January 2001 through December 2002, from a low-volume urban emergency medical services system, 185 persons presented with CVA signs and/or symptoms. Data collected included patient chief complaint, neurologic examination, patient age, vital signs, ambulance field times, patient past medical history, and blood glucose measurement with resulting prehospital interventions, efficacy of interventions, and iatrogenic complications. Five persons (2.70%), all medication-controlled diabetics, were found to be hypoglycemic. After administration of intravenous dextrose 50% by rescue personnel, improvement in neurologic condition was noted in 100% of these cases. No sequelae as a result of such care occurred. No inappropriate use, point estimate ([0]/[5][0.00%-52.20%]), or unmet need, point estimate ([0]/[9495][0.00%-0.04%]), of care was noted. The data presented in this study suggest that given similar emergency medical service system characteristics, hypoglycemic patients presenting with neurologic deficits suggestive of CVAs constitute a rare event, associated with medical histories predictive of problems involving glucose homeostasis. Blood glucose measurement in persons presenting with CVA signs and/or symptoms is only necessary given the presence of history suspicious for hypoglycemia, or rescuer inability to obtain adequate patient information. Routine prehospital blood glucose measurement in patients with suspected CVA appears unnecessary.

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1. Introduction

Hypoglycemia constitutes one of the primary differential diagnosis, given the patient presenting with focal neurologic deficits [1-4]. Routine blood glucose measurement in the management of patients presenting with signs/symptoms suggestive of cerebrovascular accidents (CVAs) has thus remained an integral component of prehospital treatment protocols for over 20 years [5,6]. Surprisingly, little is documented in the medical literature to substantiate such care [7]. One of the most common and catastrophic emergencies of the central nervous system (CNS), CVA remains the third leading cause of death in adulthood [2,8]. Over 700,000 persons will suffer a CVA, or stroke, in the United States annually [1]. Given such prevalence and mortality, the importance of prehospital CVA patient care must be recognized and subjected to investigation. The present study was therefore completed to establish an epidemiologic database defining prehospital management of suspected CVAs with attention to blood glucose measure-
ment, in the hope of developing recommendations for further treatment protocols.

2. Methods

Evanston, Ill, is a metropolitan city with approximately 73,200 residents in 8.5 square miles. Using a 911 response system for emergency medical services (EMS), all calls for ambulance service are dispatched exclusively to the fire department. A prehospital care provider in a low-volume urban EMS system [7,9], the Evanston Fire Department’s (Evanston, IL) routine ambulance response for all ill and injured persons consists of an advanced life support paramedic rescue team. All patient care decisions are made in accordance with paramedic standard operating procedures for the Saint Francis Emergency Medical Services System, Saint Francis Hospital, Evanston, Ill, and existing adult advanced cardiac life support, pediatric advanced life support, and advanced trauma life support guidelines [10-14]. Using the Cincinnati Prehospital Stroke Scale [6] for patient evaluation, prehospital CVA treatment protocols consist of initial patient assessment including neurologic examination (level of consciousness and cranial/peripheral neurologic integrity), vital signs and pulse oximetry, electrocardiographic monitoring, keep-open rate intravenous lines [15], supplemental oxygen administration, blood glucose measurement, and continual patient re-evaluation for changes in neurologic/hemodynamic condition.

All paramedic responses (run reports) were retrospectively reviewed for the 24-month period from January 2001 through December 2002. All calls that involved suspected CVAs were noted. Paramedic documentation of the word “CVA/stroke” on ambulance run reports, or patient complaint/examination suggestive of CVA, that is, hemiparesis, hemiplegia, select cases of speech/visual disturbances, confusion and/or incoordination, met inclusion criteria for this study [1,2,16,17], therefore, decreasing the potential for selection bias. To minimize discrepancies in differential diagnosis [1,2], thus reducing the chance of false positives, unconscious/unresponsive persons, as well as those presenting with exclusive complaints of syncope, seizures, generalized weakness/dizziness, obvious intoxication, known hypoglycemia/diabetic emergencies, or trauma-associated neurologic deficits, were excluded from the study population. Patient chief complaint, neurologic examination (initial assessment and subsequent changes), patient age, vital signs, ambulance field times (initial response, scene, and patient-to-hospital transportation), patient past medical history and blood glucose measurement with resulting prehospital interventions, efficacy of interventions, and iatrogenic complications were noted. Hypoglycemia was defined by paramedic standard operating procedures as a blood glucose level of 60 mg/dL or less. Blood glucose measurements were made by Ames Glucometer II (Ames Division, Miles, Inc, Elkhart, Ind) (accuracy, correlation coefficient, 0.96; precision, coefficient of variation, <2%) [18], calibrated to manufacturer specifications. As electrocardiographic monitoring, keep-open rate intravenous lines, supplemental oxygen administration, blood glucose measurement, and pulse oximetry were system-mandated routine care for the treatment of all patients with CVAs, they were subsequently excluded from consideration as prehospital interventions. Ambulance initial response time was defined as occurring from receipt of a given call for paramedic assistance by 911 dispatch until arrival of rescue personnel on location at the emergency scene. To avoid biased data caused by falsely elevated values for prehospital interventions, patients, rather than individual administrations of a given medication/procedure, were used as units of observation. Point estimate (PE) of use or need of care was determined. All prehospital care was subject to review for quality assurance by both fire department and hospital EMS supervisory personnel. Data collected did not include hospital follow-up of patients to determine the absence/presence of long-term sequelae. This study was approved by the Saint Francis Emergency Medical Services System Institutional Review Board.

3. Results

A total of 9495 paramedic run reports were reviewed (one patient per report). One hundred eighty-five persons (1.95%) presented with signs and/or symptoms suggestive of CVAs (Table 1). Of these, 5 persons (2.70%), age range, 52 to 84 years (mean, 64.40 years; SD, ±13.13), all of whom had a concurrent history of medication-controlled diabetes, were found to be hypoglycemic (mean blood glucose, 33.20 mg/dL.; SD, ±7.40), Table 2. Paramedics administered intravenous dextrose 50% to all 5 patients, with improvement in neurologic condition noted in 100%

<table>
<thead>
<tr>
<th>Signs/symptoms</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemiplegia/hemiparesis</td>
<td>104</td>
</tr>
<tr>
<td>Speech/cognitive deficitsa</td>
<td>22</td>
</tr>
<tr>
<td>Visual disturbancesb</td>
<td>1</td>
</tr>
<tr>
<td>Hemiplegia/hemiparesis</td>
<td>58</td>
</tr>
<tr>
<td>with speech/cognitive deficits</td>
<td></td>
</tr>
</tbody>
</table>

a Slurred speech, expressive/receptive aphasia.
b Visual field deficits.
of cases (corrected mean blood glucose, 221 mg/dL; SD, ±96.17). No other prehospital interventions were necessary and no iatrogenic complications occurred. No other persons within the study population were found to be hypoglycemic (mean blood glucose, 137.36 mg/dL; SD, ±63.62), and no cases of unrecognized hypoglycemia were noted.

Study population age ranged from 32 to 97 years (mean, 71.96 years; SD, ±14.55). No clinically significant association between patient age and hypoglycemia could be found. Average initial response, scene, and patient-to-hospital transportation times were 4.38 minutes (SD, ±1.57), 19.59 minutes (SD, ±5.38), and 4.36 minutes (SD, ±2.07), respectively. No persons demonstrated any hemodynamic compromise, and all patients remained conscious/responsive at all times. Past medical history was documented in 100% of patients, with 155 persons (83.78%) having no risk factors for hypoglycemia, whereas 30 persons (16.22%) presented with concomitant pathology suggestive of abnormalities in glucose homeostasis (27 cases, medication-controlled diabetes; 3 cases, liver disease/cirrhosis). Presence of risk factors for hypoglycemia vs low blood glucose measurement was tabulated for statistical analysis (Table 3). No cases of inappropriate use, PE [(0)/(5) (0.00% to 52.20%)], and no cases of unmet need, PE [(0)/(9495) (0.00% to 0.04%)], of care were noted.

4. Discussion

Hypoglycemia remains an accepted cause of focal neurologic deficits [1-4]. As the main source of energy in the CNS, glucose homeostasis is critical to brain function [3,4]. When glucose levels are decreased, CNS impairment results with varied effects, including altered level of alertness, convulsions, coma, and focal neurologic deficits that may occur even without a major alteration in consciousness [3,4,10]. Hypoglycemic-mediated cortical vasospasm, structural narrowing of selective cerebral blood vessels resulting in more pronounced localized effects of hypoglycemia, and/or vulnerability of selective neurons to hypoglycemic states have been postulated as causative mechanisms behind such events [19-23]. Immediate blood glucose level determination by prehospital care providers is therefore recommended as routine practice when faced with persons presenting with signs/symptoms of CVAs [1,11]. Given the results of this study, however, such care must be subject to question in light of the pathophysiology of hypoglycemia and its prevalence within this patient population. Rather, a selective approach to blood glucose measurement in patients with suspected CVA on the basis of clinical indication must be considered, as hypoglycemia in such instances appears to be a rarely occurring event, associated with definable risk factors. Given similar patient populations and scene times, the following recommendations for measurement of blood glucose in persons presenting with suspected CVAs are therefore introduced (Table 4). Blood glucose measurement is only necessary in the presence of history suspicious for hypoglycemia, or rescuer inability to obtain adequate patient medical information.

Hypoglycemia is defined as a constellation of characteristic signs/symptoms occurring in relation to a low blood glucose level [3,4]. Hypoglycemia does not constitute an independent process, but rather, an underlying physiological abnormality must primarily exist in order that hypoglycemic states might occur. Commonly due to diabetes, other causes of hypoglycemia remain readily identified and include liver disease, alcohol intoxication, sepsis, and certain toxic ingestions [3,4]. Of the patients in this study found to be hypoglycemic, 100% had a past medical history elicited by paramedics on initial assessment, suspicious for abnormalities in glucose homeostasis (medication-controlled diabetes). No cases were noted involving hypoglycemia in the absence of a suggestive setting (Table 3). Thus, the likelihood that a given patient with apparent CVA lacking known abnormalities in glucose homeostasis will in actuality be hypoglycemic is 0.00% (95% confidence interval [CI], 0.00% to 2.35%). Should risk factors for hypoglycemia be present, the likelihood of having a low blood glucose level is 16.67% (95% CI, 5.65% to 34.72%). Such data clearly illustrates the rarity of these events and is reflective of clinical practice in general. Although rescuers were successful in obtaining patient medical histories in all instances, it must be remembered that ability to gather such information is of paramount importance in this situation. Limiting protocol application to conscious hemodynamically stable persons, as were encountered in this population, is therefore recommended.

### Table 3 Low blood glucose measurement vs risk factor presence for hypoglycemia in the patient with suspected CVA

<table>
<thead>
<tr>
<th>Risk factor for hypoglycemia&lt;sup&gt;a&lt;/sup&gt;</th>
<th>No</th>
<th>Yes</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>155</td>
<td>0</td>
<td>155</td>
</tr>
<tr>
<td>Yes</td>
<td>25</td>
<td>5</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>180</td>
<td>5</td>
<td>185</td>
</tr>
</tbody>
</table>

<sup>a</sup> No. of patients.

### Table 4 Prehospital triage criteria: blood glucose measurement in the patient with suspected CVA<sup>a</sup>

<table>
<thead>
<tr>
<th>Blood glucose measurement indicated&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Blood glucose measurement not indicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>History suspect for pathology involving glucose homeostasis</td>
<td>History negative for pathology involving glucose homeostasis</td>
</tr>
<tr>
<td>Patient medical history unavailable</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Given conscious hemodynamically stable persons.
<sup>b</sup> Only one criterion need be present.
Only 5 (2.70%) of 185 study patients were found to have a low blood glucose measurement. Hypoglycemia is the underlying process in approximately 7% of all persons with altered mental status [3]. Based on the data presented, it appears that when faced with the patient manifesting focal neurologic deficits, hypoglycemia is encountered even less frequently. In light of such findings, recommendations for universalprehospital blood glucose measurements for patients with suspected CVA cannot be supported.

Lastly, although paramedics did not document the length of time necessary to complete blood glucose measurement, given current constraints inprehospital CVA patients care emphasizing minimization of scene times/rapid hospital access, [10] as well as concerns over rescuer exposure to blood-borne pathogens [24], any recommendations resulting in the reduction of unnecessaryprehospital care/minimization of infectious risk must be entertained.

This study is limited by the small sample size, the difficulty in extrapolating results of a low-volume urban EMS system to larger settings, and by the retrospective design with potential for selection bias. Ofnote, with respect to likelihood upper confidence limits, the triage criteria presented may result in a maximum estimated 2.35% of patients having undetected hypoglycemia. A study with greater numbers of subjects might well show this percentage to be smaller. Further investigation using prospective validation and larger patient populations is therefore necessary before the findings presented can be universally applied to allprehospital situations.

5. Conclusion

When persons present with signs and/or symptoms suggestive of CVAs,prehospital care providers must consider the possibility of hypoglycemia as a causative factor. Given the results of this study, however, routine blood glucose measurement of patients with suspected CVA lacks indication, as problems involving glucose homeostasis presenting with focal neurologic deficits occur only rarely, and then can be suspected based on patient medical history. Rather, provided a similar patient population andprehospital environment, blood glucose measurement is only necessary given the presence of risk factors for hypoglycemia, or rescuer inability to obtain adequate patient historical information. Routineprehospital blood glucose measurement in patients with suspected CVA may be an unnecessary practice, lacking clinical justification. Such policies must be questioned.

Acknowledgment

The author thanks the Evanston Fire Department, Evanston, IL and Glenn E. Aldinger, MD, Beata A. Schaible, RSN, and Mary Ann Miller, RN, Department of Emergency Medical Services, Saint Francis Hospital, Evanston, Ill, for data collection. Thanks are also extended to Raymond M. Fish, MD, College of Medicine, University of Illinois, Champaign-Urbana, IL, Kathleen Schrank, MD, School of Medicine, University of Miami, Miami, Fl, and Gary Stevens, PhD, College of Medicine, University of Florida, Gainesville, FL for statistical review and comments.

References


Original Contributions

The minimum clinically significant difference in patient-assigned numeric scores for pain

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Abstract

Objective: To determine the minimum clinically significant difference (MCSD) in patient-assigned, 11-point numeric rating scale (NRS-11) scores for pain and to determine if the MCSD varied with demographic characteristics.

Methods: Eligible emergency department patients presenting with pain were asked to rate their pain on the NRS-11 every 20 minutes. Subjects compared pain intensity by choosing from the following verbal descriptor responses: “a lot more,” “a little more,” “about the same,” “a little less,” or “a lot less” pain. The MCSD was defined as the difference between scores rated “a little more” or “a little less” severe.

Results: Three hundred fifty-four subjects were enrolled. The MCSD was 1.39 ± 1.05 (95% confidence interval, 1.27-1.51). No statistically significant difference based on sex or pain etiology was noted.

Conclusions: Findings suggest that a change of 1.39 ± 1.05 (95% confidence interval, 1.27-1.51) on the NRS-11 is clinically significant when measuring pain.

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1. Introduction

It has been estimated that greater than 60% of all emergency department (ED) patients present with conditions associated with pain [1]. As clinically significant pain relief is the end point for pain management, pain reduction and measurement have become major areas of focus for the specialty of emergency medicine and for emergency medicine research [2]. Recent literature has established that oligoanalgesia in the ED is a frequent occurrence, with reasons for this debated [2-7]. A key component to the reduction of pain is its accurate measurement. Pain can be difficult to measure and assess in the ED as a patient’s experience of pain is influenced by many factors, including their medical condition; developmental level; emotional and...
cognitive status; culture; the hospital environment; family issues and attitudes; language barriers; and levels of fear and anxiety [1].

There are currently several reliable valid pain measurement tools available for use with adult patients in the setting of acute pain. Among these, the National Institutes of Health states that “patient self-report” is the most reliable indicator of the existence and intensity of pain [8].

Perhaps the most widely used and researched scale in the emergency medicine literature is the 100-mm visual analog scale (VAS). With this scale, the patient is shown a 100-mm line, marked on one end with a descriptor such as “most imaginable pain” and marked on the other end with “no pain.” The patient is then asked to mark the site on the line that corresponds to their level of pain. The provider measures the distance in millimeters from the beginning of the line to the patient’s mark. This scale has been validated [9-11]; however, the measurement where a statistical difference in the pain score becomes clinically significant has been debated [12-14]. This scale has several limitations, including the following: the patient’s pain can only be assessed in written form; two steps are required to score; and the tool itself may be rendered invalid if multiple photocopies of it are made.

Many hospital EDs currently assess adult patients’ pain using an 11-point numeric rating scale (NRS-11). With the NRS-11, patients are asked to choose one number, on a scale from 0 (no pain) to 10 (severe pain), corresponding to the intensity of their pain. Advantages of this instrument include ease of administration and scoring, multiple response options, and no age-related difficulties in using the scale [10].

The objectives of this study were to determine the minimum clinically significant difference (MCSD) in NRS-11 pain scores and to determine if this difference varies with age, sex, ethnicity, primary spoken language, or etiology of the patient’s pain (traumatic vs nontraumatic).

2. Methods

2.1. Study design

This study was a prospective descriptive trial examining the MCSD in patient-assigned NRS-11 scores for pain. The investigation was approved by the Maine Medical Center Institutional Review Board.

2.2. Setting and study population

The Department of Emergency Medicine at Maine Medical Center is an academic referral center with an emergency medicine residency program. Maine Medical Center’s annual ED census was approximately 53,000 patients at the time of the study.

All patients presenting to the Maine Medical Center Department of Emergency Medicine with acute pain who were able to give written informed consent were eligible for study inclusion. Children aged 8 years or older who were able to give assent were included. Those patients who were unable to speak English and/or read the English consent document were excluded. There was no upper age limit for inclusion in the investigation. Subjects were excluded if they left the ED before being evaluated by a physician.

2.3. Study protocol

The study was conducted during 8-hour shifts on 50 nonconsecutive days spanning from September 2003 to April 2004. All days of the week and shifts (day, evening, and night) were included. All patients presenting to the ED with a chief complaint of any type of pain were approached and screened for study inclusion by a study investigator or trained research assistant. Informed consent or assent was obtained. At enrollment, subjects were asked to rate their pain intensity using the 11-point NRS. After 20 minutes, the subjects were asked again to indicate their pain intensity and then were asked to contrast their current intensity with the pain intensity at the previous assessment. Subjects were not allowed to refer to their prior written pain intensity score. This process was repeated every 20 minutes for 2 hours, or until the patient reported no pain or left the ED, yielding 6 contrasts for each subject. Study-related pain assessments began after the patient had been evaluated by a physician. Pain scores of 5 or greater and requests for pain medication were reported to the patient’s primary nurse and/or physician as NRS-11 scores of 5 to 6 are considered indicative of moderate pain [15].

Subjects were instructed that there was no correct or incorrect response and that their own judgments of pain intensity would likely differ from those of other patients. Upon completion of the pain intensity estimates and contrasts, all subjects were informed of the study objective. Additional demographic information on each patient including age, sex, race, primary spoken language, pain score at triage, chief complaint, medications given, discharge

![Fig. 1 11-Point numeric rating scale for pain.](image)
diagnosis, and patient disposition was collected by the enrolling investigator or research assistant.

2.4. Measurement

2.4.1. 11-Point numeric rating scale

A 0 to 10 NRS (NRS-11 scores, Fig. 1) labeled with the descriptors “no pain” and “worst pain you can imagine” was used by subjects to rate their pain level.

2.4.2. Comparison scale

After assigning an NRS-11 score to their pain, participants were asked to compare their current pain state with the pain they were experiencing when they chose their previous pain score. Each participant was asked to choose one of the following descriptors: “much more pain,” “a little more pain,” “about the same pain,” “a little less pain,” or “much less pain.”

2.5. Data analysis

For each paired contrast, the difference between the two pain scores and the comparison scale descriptor was recorded. The MCSD was defined as the difference in mean pain scores for pairs rated either “a little less pain” or “a little more pain.” Data were analyzed using SPSS 11.0 (SPSS, Inc, Chicago, Ill) statistical software. Descriptive statistics including mean, SD, and 95% confidence intervals (CIs) were used. The independent samples t test was used to compare mean scores between groups.

3. Results

During the investigation, informed consent was obtained from 356 patients. Two patients were identified as having left the ED before completing their evaluation, leaving 354 subjects who completed the study protocol for final data analysis. Eighty-nine (25.2%) subjects were enrolled on weekends, whereas the remaining 265 (74.8%) subjects were enrolled on weekdays. Subjects were enrolled during all 3 shifts, with 163 (46.0%) enrolled on the day shift, 116 (32.8%) on the evening shift, and 75 (21.2%) on the night shift. Table 1 describes the characteristics of the study subjects.

A total of 1515 pain comparisons were made. Of these comparisons, 244 were rated as “a little less pain” and 94 were rated as “a little more pain.” The mean difference between current and preceding NRS-11 scores in these 338 comparisons of interest was $1.39 \pm 1.05$ (95% CI, 1.27-1.51). Mean differences in pain scores by comparison category are reported in Table 2.

When comparing NRS-11 scores according to sex, the MCSD for females was $1.38 \pm 0.88$ (95% CI, 1.25-1.50) and for males $1.39 \pm 1.05$ (95% CI, 1.27-1.51). This difference was not found to be statistically significant ($t = -0.233, df = 306, P = .824$).

No statistically significant difference in the MCSD based on a traumatic or nontraumatic pain etiology was found ($t = 0.891, df = 206, P = .374$), with the MCSD for traumatic pain etiology being $1.48 \pm 1.06$ (95% CI, 1.25-1.70) and for nontraumatic etiology being $1.36 \pm 1.05$ (95% CI, 1.22-1.49).

There were too few subjects younger than 18 years (n = 13), of other-than-white race (n = 19), or non–primary English speakers (n = 8) to compare the MCSD for different ages, races, and primary languages.

A triage nurse’s pain assessment using the NRS-11 was noted for 325 (91%) subjects, with the mean score at triage being $6.60 \pm 2.58$ (95% CI, 6.31-6.88). Triage NRS-11 scores ranged from 0 to 10. Fig. 2 displays the frequency with which the range of scores was reported. Of all the subjects reporting pain at triage, 166 (46.9%) received a pharmacological intervention while in the ED (ie, nitroglycerine for treatment of chest pain, prochlorperazine for treatment of migraine headache). One hundred nineteen

### Table 1

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>148 (41.8)</td>
</tr>
<tr>
<td>Female</td>
<td>206 (58.2)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>4 (1.1)</td>
</tr>
<tr>
<td>Black</td>
<td>9 (2.5)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>6 (1.7)</td>
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<tr>
<td>White</td>
<td>335 (94.6)</td>
</tr>
<tr>
<td>Age (y)</td>
<td></td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>41 (52-30)</td>
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<tr>
<td>Primary language</td>
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<tr>
<td>Amharic</td>
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<td>Somali</td>
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<td>Spanish</td>
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<tr>
<td>Nontraumatic</td>
<td>231 (65.3)</td>
</tr>
<tr>
<td>Disposition</td>
<td></td>
</tr>
<tr>
<td>Discharged</td>
<td>289 (81.6)</td>
</tr>
<tr>
<td>Admitted</td>
<td>61 (17.2)</td>
</tr>
<tr>
<td>AMA</td>
<td>2 (0.6)</td>
</tr>
<tr>
<td>Operating room</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Transferred</td>
<td>1 (0.3)</td>
</tr>
</tbody>
</table>

IQR indicates interquartile range; ASL, American Sign Language; AMA, patient left against medical advice.

### Table 2

<table>
<thead>
<tr>
<th>Comparison category</th>
<th>n</th>
<th>mean (SD)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Much less pain</td>
<td>96</td>
<td>4.47 (2.67)</td>
<td>3.93-5.01</td>
</tr>
<tr>
<td>Little less pain</td>
<td>244</td>
<td>1.46 (.958)</td>
<td>1.34-1.58</td>
</tr>
<tr>
<td>About the same pain</td>
<td>1045</td>
<td>0.8 (.374)</td>
<td>0.06-0.10</td>
</tr>
<tr>
<td>Little more pain</td>
<td>94</td>
<td>1.23 (1.16)</td>
<td>1.01-1.46</td>
</tr>
<tr>
<td>Much more pain</td>
<td>36</td>
<td>2.82 (1.90)</td>
<td>2.17-3.47</td>
</tr>
</tbody>
</table>
(39.53%) subjects received an analgesic medication while in the ED. One hundred eighty-eight (53.1%) subjects reporting pain received no pharmacological intervention while in the ED. For these subjects, the mean NRS-11 score reported at triage was 5.93 ± 2.47 (95% CI, 5.54-6.31).

4. Discussion

This investigation found the MCSD in NRS-11 pain scores to be approximately 1.39, consistent with the findings of several similar studies of both the NRS and VAS. Todd [16] evaluated acute pain in ED patients using the VAS on admission to the ED, with repeated measures every 20 minutes for the next 2 hours. Patients were asked at each subsequent measurement to compare their current pain with the pain at the previous measurement. Todd [16] concluded the minimum clinically significant change in VAS score to be 13 mm. Similarly, Kelly [17] found the minimum clinically significant change in VAS score to be 9 mm and found no statistically significant difference between sex, age, or pain etiology.

Berthier et al [18] compared the NRS-11, the VAS, and a 5-point verbal rating scale in 290 consecutive ED patients admitted with acute pain. They found that the VAS and NRS-11 correlated closely for patients with traumatic and nontraumatic pain. They could not use the VAS with 19.5% of trauma patients nor with 11% of subjects with nontraumatic pain. In contrast, the NRS-11 was successfully used in 96% of all subjects. In subjects with traumatic pain, the NRS-11 was deemed more reliable. If the pain was nontraumatic in nature, the NRS-11 was equivalent to the VAS. Given these findings, Berthier et al [18] concluded that the NRS-11 is an appropriate and reliable scale for use in EDs.

In a recent study, Bijur et al [19] validated the verbally administered NRS-11 in the ED setting and compared it with the VAS in measuring acute pain. They studied 108 adult patients presenting to an inner-city ED in acute pain. They found the MCSD for the NRS-11 to be 1.3 and 1.4 for the VAS. They also concluded that the verbally administered NRS-11 could be substituted for the VAS in acute pain management.

In addition to evaluating these pain measurement tools in ED patients, several authors have evaluated the VAS and NRS using clinician-assigned scores. Todd and Funk [13] assessed the MCSD in VAS scores assigned by physicians. In this study, physicians were shown written scenarios describing patients in moderate to severe pain. They were asked to rate their perception of the patient’s pain on a 100-mm VAS and then asked to compare the perceived pain with the pain of the patient in the previous scenario. The authors concluded the mean MCSD to be 18 mm. Strout and Burton [14] used a similar methodology to determine the MCSD in NRS-11 scores assigned by physicians. They found this difference to be 1.45 in scenarios describing adult and pediatric patients presenting to the ED in acute pain [14].

In some EDs, the NRS-11 is used to assess pain in children as well as in adult patients. Powell et al [20] studied the VAS in children aged 8 to 15 years and determined the MCSD in NRS-11 pain scores for this population to be 10 mm. Because of the small number of pediatric patients enrolled in our investigation, we were unable to determine the MCSD in children using the NRS-11.

We found that a change of approximately 1.39 on the NRS-11 for assessing pain is clinically important for patients. This information may be helpful for emergency practitioners to more accurately assess pain and evaluate the results of various treatments aimed at decreasing pain in ED patients.

Interestingly, we found that although 71.8% of subjects reported pain scores of 5 or greater at triage, only 46.9% of subjects received any type of pharmacological intervention for their pain. This number includes those who received an analgesic and nonanalgesic medication, such as those receiving nitroglycerin for chest pain. Our investigation did not record nonpharmacological measures aimed at pain reduction, such as the application of ice or the elevation of injured extremities; however, we hypothesize that the number of nonpharmacological interventions was probably relatively small and isolated to those subjects with traumatic injuries as study staff rarely recalled witnessing such interventions. Despite recent increased attention to pain related issues in the ED, these data indicate that many patients are likely receiving less than ideal attention to their pain experiences.

4.1. Limitations and future questions

There are several important limitations to our study. Because of the availability of investigation staff, subjects were not enrolled consecutively, but as a convenience sample. In an attempt to minimize bias introduced by the convenience sample, enrollment times and days were chosen to provide a sampling of all shifts and days of the week.
While Maine Medical Center serves a diverse population, small numbers of children, non-white race, and non-primary English speaking subjects were included in this sample. In attempting to discern the reasons for this, it was found that although many potential subjects could speak English as their secondary language, enrolling investigation staff frequently felt that the patient could not fully participate in the informed consent process because of their inability to read and fully comprehend the English language informed consent document. In addition, we believe that fewer children than expected were enrolled because, although many children experience pain in the ED, pain is less frequently their chief complaint (our flag to screen for enrollment eligibility) than in their adult counterparts.

Future studies are needed to specifically assess clinically significantly changes in pain scores for children and patients of non-white and non-primary English speaking populations. Researchers should consider translating informed consent documents into languages frequently spoken in the populations they serve to increase enrollment in diverse populations.

4.2. Conclusions

In this cohort, the MCSD in NRS-11 pain scores was 1.39 ± 1.05 (95% CI, 1.27-1.51). We identified no statistically significant difference in the MCSD based on subject sex or etiology of pain and were not able to evaluate differences based on age, ethnicity, or primary spoken language. These results can assist in guiding clinicians who treat pain, as well as in the planning and interpretation of future pain research.

Acknowledgments

The authors gratefully acknowledge the assistance Beth Villandry, MD, Kimberly Perreault, DO, Cecily Cannon, MS, and Jennifer Lee, MD, provided in collecting data. The authors thank Michael Baumann, MD, for his thoughtful comments in the editing of final versions of this manuscript.

References


Original Contributions

Fluid resuscitation of trauma patients: how fast is the optimal rate?

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Accepted 16 March 2005

Abstract The Advanced Trauma Life Support guidelines recommend an initial rapid infusion of fluid (1-2 L) in trauma and hemorrhage victims as a diagnostic procedure to aid treatment decisions. Although patient response to initial fluid resuscitation is the key to determining therapeutic strategies, the appropriate rate of infusion is not clearly defined. Ninety-nine adult (age >16 years) blunt trauma victims with hypotension were enrolled. Patients were classified into 3 groups according to hemodynamic state after initial fluid resuscitation and requirement of surgical intervention. Total volume and rate of infusion differed significantly between the groups (P < .05). Patients requiring fluid administration at higher rate were all hemodynamically unstable and required immediate surgical intervention. Moreover, rate of infusion was the best predictor of the patients who required immediate surgical intervention. Moderate fluid infusion rate should be considered to allow identification of the patient’s response to initial fluid resuscitation.

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1. Introduction

Despite major advances in the management of trauma victims, traumatic injury remains one of the leading causes of death during the first 3 decades of life [1]. Moreover, effective treatment of critically injured patients with hypovolemic shock continues to be a formidable challenge. The main management strategies in hemorrhagic shock are the arrest of bleeding and replacement of circulating fluid volume. The Advanced Trauma Life Support guidelines recommend an initial rapid infusion of fluid in the management of trauma and hemorrhage victims as a diagnostic procedure to aid in further treatment decisions [2]. Patients still unstable after aggressive volume resuscitation must undergo urgent surgery to control the source of bleeding.

Although patient response to initial fluid resuscitation is the key to determining an appropriate therapeutic strategy, the rate of fluid infusion has not been clearly defined. Moreover, recent controversies have arisen regarding end points in fluid resuscitation [3]. Several authors suggest that aggressive fluid resuscitation before control of bleeding may result in increased blood loss [4,5]. However, hypotensive patients should be stabilized rapidly with moderate fluid infusion to achieve and maintain perfusion of essential organs. Both inadequate and excessive fluid resuscitation
Hypotension after Blunt Trauma (n = 99)

Initial fluid resuscitation

Hemodynamically stable

Hemodynamically unstable

SBP < 90 mmHg or HR >120

Group A (n = 33)

Group B (n = 27)

Group C (n = 39)

Non surgical intervention

Surgical intervention

Fig. 1 Initially, patients were classified into 3 groups according to hemodynamic parameters after initial fluid resuscitation and surgical intervention. Hemodynamically unstable was defined as sustained hypotension (SBP <90 mm Hg) or prolonged tachycardia (HR >120 beats per minute), with no or unsatisfactory response to initial fluid resuscitation.

2. Material and methods

Adult (age ≥16 years) blunt trauma victims with systolic arterial blood pressure (SBP) of 90 mm Hg or less upon admission were identified for retrospective analysis. All patients included in the study were initially evaluated and managed at our institute. Patients who were dead on arrival or not admitted directly to our hospital were excluded from the study. Standard trauma resuscitation protocols were used for all other components of care. None of the patients had undergone prehospital administration of intravenous fluids.

If shock was present, at least two large-bore percutaneous catheters were placed, and a rapid bolus infusion of Ringer’s lactate was started immediately. Central venous access was used as necessary. If a hemodynamically normal state was achieved, the rate of fluid infusion was reduced and adjusted to maintain adequate vital signs. If the patient remained unstable, further rapid fluid and blood infusions were performed. Data on total fluid volume and time of initial resuscitation until surgical intervention or transfer to the intensive care unit (ICU) were collected for each patient. All surgical interventions to control hemorrhage were reviewed and reevaluated. The clinical outcome, Injury Severity Score (ISS), and predicted probability of survival of each study patient were obtained from medical records and the trauma registry.

Table 1 Characteristics of the study patients

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>33</td>
<td>27</td>
<td>39</td>
</tr>
<tr>
<td>Age (y)</td>
<td>47.6 ± 19.2</td>
<td>45.9 ± 20.5</td>
<td>51.5 ± 21.5</td>
</tr>
<tr>
<td>Initial SBP (mm Hg)</td>
<td>79.8 ± 11.0</td>
<td>71.5 ± 13.5</td>
<td>63.7 ± 20.4***</td>
</tr>
<tr>
<td>Initial HR (beats per minute)</td>
<td>90.2 ± 27.7</td>
<td>101.2 ± 25.8</td>
<td>111.6 ± 135.2***</td>
</tr>
<tr>
<td>ISS</td>
<td>22.1 ± 13.6</td>
<td>28.8 ± 15.4</td>
<td>39.7 ± 13.1***</td>
</tr>
<tr>
<td>RTS</td>
<td>6.0 ± 1.7</td>
<td>5.7 ± 1.5</td>
<td>3.7 ± 2.2***</td>
</tr>
<tr>
<td>TRISS</td>
<td>0.74 ± 0.29</td>
<td>0.71 ± 0.31</td>
<td>0.29 ± 0.29***</td>
</tr>
<tr>
<td>Mortality</td>
<td>2 (6.1%)</td>
<td>3 (11.1%)</td>
<td>32 (82.1%)***</td>
</tr>
</tbody>
</table>

* P < .05 compared with A.
** P < .05 compared with B.
Data are presented as mean ± SD. Differences between groups were considered to be statistically significant at $P < .05$, as determined by 1-way analysis of variance, Tukey test, or $\chi^2$ and Fisher exact tests.

3. Results

Ninety-nine patients were entered into the study during a period of May 1999 through October 2002. There were 66 men and 33 women. Mean age was 48.6 ± 20.4 years. The causes of injury included motor vehicle crash (n = 31), fall (n = 29), pedestrian-automobile accident (n = 32), and other events (n = 7).

3.1. Patient groups

Initially, patients were classified into 3 groups according to hemodynamic parameters after initial fluid resuscitation and surgical intervention (Fig. 1): patients who were hemodynamically stable after initial fluid resuscitation and required no further surgical intervention (group A, n = 33); patients who were hemodynamically stable after initial fluid resuscitation but required surgical intervention to control hemorrhage (group B, n = 27); and patients who were hemodynamically unstable after initial fluid resuscitation and required further fluid resuscitation and immediate surgical intervention (group C, n = 39). Hemodynamic instability was defined as sustained hypotension (SBP <90 mm Hg) or prolonged tachycardia (heart rate [HR] >120 beats per minute) with absent or unsatisfactory response to initial fluid resuscitation. Surgical intervention included radiological transarterial embolization to control bleeding in patients with pelvic fracture.

3.2. Clinical characteristics

The demographic and outcome comparisons are listed in Table 1. Initial SBP decreased significantly in group C in comparison with that in other groups. Also, initial HR was significantly higher in group C than in group A or B. The ISS was significantly higher in group C than that in group A or B. Revised Trauma Score (RTS) and TRISS were significantly lower in group C that in group A or B. However, there was no significant difference between group A and B in initial SBP, ISS, RTS, or TRISS. Actual mortality rates were 6.1% in group A, 11.1% in group B, and a significantly high 82.1% in group C.

The sites of hemorrhage in each group are shown in Table 2. Multiple hemorrhage sites were present in 20.5% of patients in group C.

3.3. Hemodynamic response to initial fluid resuscitation

Patients in groups A and B responded to initial fluid resuscitation. Systolic blood pressure was restored 79.8 ± 11.0 to 122.1 ± 15.7 mm Hg in group A and 71.5 ± 13.5 to 126.3 ± 15.7 mm Hg in group B, and HR decreased 90.2 ± 27.7 to 85.7 ± 7.5 beats per minute in group A and 101.2 ± 25.8 to 94.1 ± 14.5 beats per minute in group B (Fig. 2). However, patients in group C failed to respond to fluid resuscitation; marked tachycardia (111.6 ± 35.2 to 113.5 ± 25.2 beats per minute) with depressed SBP (63.7 ± 20.4 to 77.2 ± 16.1 mm Hg) continued even after initial resuscitation (Fig. 2).

3.4. Fluid resuscitation

Initial resuscitation time between admission and surgical intervention in groups B and C was significantly shorter than the time between admission and transfer to the ICU in group A (Table 3). However, there was no significant

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Site of hemorrhage or cause of hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
</tr>
<tr>
<td>Hemorrhage</td>
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<tr>
<td>Pleural</td>
<td>1</td>
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<td>Peritoneal</td>
<td>2</td>
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<td>Retroperitoneal</td>
<td>3</td>
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<td>External hemorrhage</td>
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<td>Multiple extremity fracture</td>
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<tr>
<td>Multiple sites</td>
<td></td>
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<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>Neurogenic shock</td>
<td>5</td>
</tr>
<tr>
<td>No identifiable cause</td>
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</table>

Fig. 2 Hemodynamic response to initial fluid resuscitation. Patients in groups A and B responded to initial fluid resuscitation; SBP was restored and HR was decreased. However, patients in group C failed to respond to fluid resuscitation; they remained in marked tachycardia with depressed SBP. OA indicates on admission; ER, end of initial resuscitation.
difference in initial resuscitation time between group B and C. Total fluid administered during the initial resuscitation differed significantly between all groups. Also, a greater total amount of fluid and packed red blood cell transfusion (PRBC) were administered to group C patients than to group A or B patients during the first 24 hours after injury (Table 3). There was a significant difference between all groups in the total amount of fluid and PRBC administered during the first 24 hours.

Hemodynamically stable patients did not require fluid administration at more than 60 mL/min during the initial resuscitation (Fig. 3). Patients requiring fluid administration at higher rate were all hemodynamically unstable and required surgical intervention (Fig. 3).

### 3.5. Receiver operating characteristic curves

We constructed receiver operating characteristic (ROC) curves (derived from rate of infusion, ISS, RTS, and initial SBP) and compared the areas under the ROC curves to predict a group C patient. Receiver operating characteristic curves indicated that rate of infusion (the area under the ROC curve, 0.95 [95% confidence interval, 0.92-0.99]) was the best predictor of group C patients characteristics (Fig. 4). On the ROC curves, the cutoff value for the rate of infusion fluid (the value closest to the upper left corner of the ROC plot) was 45 mL/min (sensitivity, 79.5%; specificity, 91.7%).

### 4. Discussion

Although early, rapid fluid resuscitation remains the cornerstone of treatment for trauma and hemorrhage victims, the most important management principle in treating hemorrhagic shock is to find the source of blood loss and stop it [6,7]. Patient response to initial fluid resuscitation should be observed to identify those patients with ongoing bleeding requiring surgical control [2,8]. It is generally accepted that 3 response patterns can be identified to initial fluid administration in hemorrhagic shock: rapid responders, transient responders, or minimal or nonresponders [2,8]. Nonresponders fail to show any hemodynamic improvement after fluid administration because of ongoing hemorrhage at a greater rate. These patients need immediate surgical intervention.

| Table 3  Fluid volumes and times of the resuscitation |
|---------------------------------|----------|----------|
| A                               | B        | C        |
| Initial fluid volumes (mL)      | 1488 ± 1136 | 2612 ± 1329* | 4061 ± 1979*** |
| Initial resuscitation time (min) | 98.6 ± 39.3 | 77.4 ± 32.1* | 65.5 ± 27.8** |
| 24 h Fluid (mL)                 | 6284 ± 2757 | 13400 ± 7133* | 20053 ± 9745*** |
| 24 h PRBC (U)                   | 2.1 ± 3.7 | 12.8 ± 12.6* | 38.4 ± 23.3** |

Initial resuscitation time indicates time from admission to until surgical intervention (group B and C) or from admission to until transfer to ICU (group A); 24 h Fluid, intravenous fluid administration during the first 24 hours; 24 h PRBC, packed red blood cell transfusion during the first 24 hours.

* $P < .05$ compared with A.

** $P < .05$ compared with B.
rather than volume replacement to control the hemorrhage. Otherwise, the prognosis is poor.

Concern has been expressed about aggressive fluid resuscitation before control of bleeding. Some investigators argue that it may disrupt thrombus formation, increase bleeding, and decrease survival [3-5,9]. Restricted fluid resuscitation may have a positive effect on uncontrolled hemorrhage but a negative effect on tissue perfusion in shocked patients who respond to fluid with stabilization of their vital signs. Therefore, it appears that a rational, moderate fluid infusion rate should be considered to allow identification of the patient’s response to initial fluid resuscitation.

The result indicated that patients requiring fluid infusion at more that 60 mL/min were all hemodynamically unstable and required immediate surgical intervention. These patients were considered nonresponders who needed immediate surgical intervention to control hemorrhage rather than aggressive fluid resuscitation. Interestingly, increasing the fluid administration at higher rate did not produce hemodynamic stability. Aggressive fluid resuscitation at higher rate may result only in excessive fluid administration and hemodilution in cases of uncontrolled hemorrhage. Receiver operating characteristic curves showed that the rate of infusion was the best predictor of whether uncontrolled hemorrhage requiring urgent surgical intervention would occur in our hypotensive patients rather than initial SBP, ISS, or RTS. On the ROC curves, the cutoff value for the rate of infusion fluid (the value closest to the upper left corner of the ROC plot) was 45 mL/min (sensitivity, 79.5%; specificity, 91.7%). Therefore, 45 mL/min might be the fluid administration rate at which the type of response to initial resuscitation can be identified in trauma victims with hypotension. It would take 20 to 45 minutes for an adult patient to receive the standard 1-2 L crystalloid infusion recommended by the Advanced Trauma Life Support guidelines at this rate.

Lewis [10] developed a computer simulation model to evaluate prehospital fluid administration and found that fluids would be beneficial to increase blood pressure only if the bleeding rate was moderate and the infusion rate was approximately equal to it. He suggested that bleeding rates greater than 100 mL/min or less than 15 mL/min would not be affected by intravenous therapy [10]. Our findings agree with previous findings indicating that a rapid infusion rate does not restore blood pressure if the concomitant bleeding is occurring at a greater rate.

The fact that the rate of fluid administration was not always consistent throughout the resuscitation could be considered a limitation to this study. The rate may have been inconsistent for patients in groups A and B, who achieved a hemodynamically normal state after initial resuscitation, because the rate of fluid administration was reduced after the initial bolus infusion. However, the rate of fluid administration in the group C patients was consistent during the initial resuscitation because the rate of infusion could not be reduced because of hemodynamic instability.

It should be noted that group C patients had a high mortality rate in this study. The predicted survival rate (TRISS) in group C was low enough to explain the high mortality rate; however, the mortality rate can be reduced in such patients if the time between initial resuscitation and surgical intervention was decreased. It is undoubtedly that an initial imaging assessment (chest x-ray, pelvic x-ray, and abdominal ultrasonography) provides useful information to detected sources of hemorrhage. Nonetheless, identifying the rate of infusion required also could be a good strategy for determining whether immediate surgical intervention is indicated. Further studies are required to evaluate this possibility and to determine the role of initial fluid infusion rate in the management of trauma and hemorrhage victims.

In summary, our findings show that increasing the fluid administration rate did not produce hemodynamic stability. Aggressive fluid resuscitation at a higher rate may result in excessive fluid resuscitation and may cause a poor outcome in patients. The rate of infusion was the best predictor of whether uncontrolled hemorrhage would occur in our hypotensive patients.

References

Original Contributions

Trends in the incidence of carbon monoxide poisoning in the United States

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Accepted 23 March 2005

Abstract

Purpose: Recent data demonstrate that the mortality rate from carbon monoxide poisoning has declined over the past 2 decades. It is not known whether this decrease in mortality is reflective of the total burden of carbon monoxide poisoning. This study sought to examine trends in other potential indicators of the incidence of carbon monoxide poisoning in the United States.

Basic Procedures: Published data from US poison control centers (PCCs) were used to calculate annual rates of calls regarding carbon monoxide exposures. Data on numbers of carbon monoxide–poisoned patients treated with hyperbaric oxygen (HBO) were used to calculate annual treatment rates. Trends in rates of carbon monoxide–related mortality, calls to PCCs, and HBO treatment were then compared.

Main Findings: Contrary to the decline in carbon monoxide–related mortality from 1968 to 1998, rates of calls to PCCs significantly increased over the same period. Neither rates of PCC calls nor HBO treatment changed significantly from 1992 to 2002. The latter 2 measures were strongly correlated.

Principal Conclusions: Although deaths from carbon monoxide poisoning have clearly decreased in the United States, other indicators of the incidence of the condition suggest that the total burden (fatal and nonfatal) may not have significantly changed. Efforts to prevent carbon monoxide poisoning should not be relaxed.

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1. Introduction

Carbon monoxide poisoning is common in the United States, accounting for an estimated 40,000 to 50,000 ED visits annually [1]. Exposure to carbon monoxide can be either intentional (suicidal) or unintentional (accidental). It is generally believed that unintentional carbon monoxide poisoning may be partially preventable through public education programs and/or governmental regulations. In support of this concept, a recent study reported a decline in carbon monoxide–related mortality rates from 20.2 to 8.8 deaths per million person years during the period 1968 to 1998 [2]. Most of this reduction occurred in unintentional motor vehicle–related carbon monoxide poisoning deaths and correlated with the introduction of the catalytic converter to automobiles in 1975.

Although trends such as these may suggest that the problem of carbon monoxide poisoning has been solved, it should be recognized that such data represent only fatal episodes. Because nonfatal carbon monoxide poisoning can result in significant long-term morbidity even when treated appropriately [3], this study was conducted to determine...
whether declining death rates are reflective of all forms of carbon monoxide poisoning. To do this, trends in 2 other potential indicators of the disease burden in the United States, specifically (1) calls to poison control centers (PCCs) regarding cases of carbon monoxide exposure and (2) national rates of hyperbaric oxygen (HBO) treatment of carbon monoxide poisoning, were examined and compared.

2. Methods

Summary information on calls to US PCCs are published yearly in the American Journal of Emergency Medicine as the Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. Reports containing data from 1985 to 2002 were searched for the total number of calls received regarding cases of carbon monoxide exposures [4-21]. Participating centers also report the size of the population that they serve, allowing calculation of the rate of calls for carbon monoxide exposure per million person population per year.

Information on the number of patients treated with HBO for carbon monoxide poisoning was collected in a recent survey of US HBO treatment facilities [22]. In that survey, treatment data for the years 1992 to 2002 were requested from the 320 known hyperbaric facilities in the country, with responses obtained from 310. Full methodological details are available in that publication. For the present comparison, annual HBO treatment rates were calculated by dividing the total number of yearly treatments by US Census Bureau population estimates, using the national resident midpoint population estimates for each year [23,24].

For each of the 2 data sets, linear regression was used to calculate a best-fit line through the annual rates and then to determine whether the slope was significantly different than zero, which would indicate a positive or negative change over the period studied. Comparison of trends in annual rates for poison center calls and HBO treatment for the overlapping years of 1992 to 2002 was performed using linear regression analysis.

3. Results

Annual call rates to US PCCs regarding cases of carbon monoxide exposure are displayed in Fig. 1A. Call rates rose from 31.1 per million persons served in 1985 to 95.4 per million persons served in 1996 and then fell to 54.5 per million persons served in 2002. Over the entire 18 years, there was a significant increase in the rate of calls (\( P = .0022 \)). From 1985 to 1998, years for which mortality rate data are available, there was an increase in call rate from 31.1 to 67.5 per million persons served (\( P < .0001 \)). From 1992 to 2002, years for which comparative hyperbaric treatment data are available, there was no significant change in the rate of calls (\( P = .3975 \)).

Hyperbaric oxygen treatment rates for carbon monoxide poisoning rose from 1992 to 1995, then generally declined from 1995 to 2002, as shown in Fig. 1B. Similar to poison center calls for the period, there was no significant overall change in the rate of hyperbaric treatment (\( P = .1946 \)). However, rates of calls to PCCs and rate of hyperbaric treatment of carbon monoxide poisoning were strongly correlated (\( r = 0.8209, P = .0020 \) (Fig. 2).

4. Discussion

Mortality from carbon monoxide poisoning has declined in the past 2 decades, driven largely by a reduction in deaths
perbaric oxygen is generally used to treat the most severely poisoned subset of patients [25]. It has been estimated that approximately 6% of carbon monoxide–poisoned patients seen in EDs nationally are treated with HBO [1]. As long as the spectrum of severity of the condition and treatment practices has not been recognized to have changed significantly during the period surveyed, the national rate of HBO treatment should serve as a qualitative marker of total disease incidence.

As is seen in Fig. 1B, hyperbaric treatment rates fluctuated over the decade studied but did not significantly change overall. Furthermore, the strong correlation between poison center calls and hyperbaric treatment rates suggest that both are valid indicators of the same group of patients, those with nonfatal carbon monoxide poisoning.

It would thus appear that the declining death rate from carbon monoxide poisoning might not tell the entire story. Two indicators of nonfatal poisoning have not significantly changed in recent years. Because fatalities represent only a small fraction of the total poisoned population, it is not unreasonable to speculate that the total number of individuals poisoned with carbon monoxide nationally may not have significantly decreased in the past 2 decades. A declining mortality rate from the condition is very laudable, but it should not be used as evidence that efforts to prevent carbon monoxide poisoning can be relaxed.

References

Incidence of carbon monoxide poisoning in the United States 841


Original Contributions

Comparison of racemic albuterol and levalbuterol in the treatment of acute asthma in the ED

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Accepted 8 April 2005

Abstract

Background: Acute asthma is often treated with racemic albuterol, a 1:1 mixture of (R)-albuterol and (S)-albuterol. Levalbuterol is the single-isomer agent comprised (R)-albuterol, an active bronchodilator, without any effects of (S)-albuterol.

Objective: To compare emergency department (ED) admission rates of patients presenting with acute asthma who were treated with either racemic albuterol or levalbuterol.

Setting: Suburban community teaching hospital.

Design: Retrospective observational case review.

Methods: Emergency department patients presenting with acute asthma at 2 different sites were reviewed over 9- and 3-month consecutive periods. Outcome measures included ED hospital admission rate, length of stay, arrival acuity, and treatment costs. Patients were excluded if younger than 1 year or if no treatment of acute asthma was rendered.

Results: Of the initial 736 consecutive cases, significantly fewer admissions (4.7% vs 15.1%, respectively; \( P = .0016 \)) were observed in the levalbuterol vs racemic albuterol group. Of the subsequent 186 consecutive cases, significantly fewer admissions were also observed (13.8% vs 28.9%, respectively; \( P = .021 \)) in the levalbuterol vs racemic albuterol group. Treatment costs were lower with levalbuterol mainly because of a decrease in hospital admissions.

Conclusion: Levalbuterol treatment in the ED for patients with acute asthma resulted in higher patient discharge rates and may be a cost-effective alternative to racemic albuterol.

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1. Introduction

Asthma is one of the most common illnesses treated in the emergency department (ED) setting. It is reported that nearly 15 million Americans have asthma, and the more than 2 million annual ED visits result in costs in excess of $6 billion with hospitalizations accounting for the largest portion of these costs [1-4]. In the United States from 1992 to 1999, the absolute number and rate of ED visits for asthma increased by 36% and 29%, respectively [4]. An evaluation of 1,448,555 consecutive patients presenting to 15 EDs in northern New Jersey from 1997 to 1999 demonstrated an increasing number of asthma visits (Fig. 1). In addition, an increasing proportion of asthma-related hospital admissions from the ED was also reported. In the calendar year 1999, it was shown that 2.7% of all ED admissions were due to acute asthma and the average acute asthma admission rate from 1997 to 1999 was 16.4% across the 15 northern New Jersey EDs studied (Emergency Medical Associates, Livingston, NJ).

Multiple therapeutic modalities are available to the ED physician to manage acute asthmatic episodes, including $\beta_2$-adrenergic agonists and corticosteroids, which are the mainstays of therapy. The most widely prescribed therapy for asthma in the inpatient, outpatient, and ED settings are the $\beta_2$-adrenergic agonists. Human $\beta_2$-adrenergic receptors are specifically designed to allow a conformational fit with L-epinephrine, the (R)-stereoisomer. Synthetic $\beta_2$-adrenergic agonists such as racemic albuterol structurally mimic epinephrine and bind to the $\beta_2$-adrenergic receptor site [5]. However, the most commonly used $\beta_2$-adrenergic agonist, is a 1:1 mixture of (R)- and (S)-albuterol stereoisomers. (R)-albuterol, also known as albuterol, solely binds to the $\beta_2$-adrenergic receptor, producing bronchodilation, whereas (S)-albuterol, because of its structural conformation, does not effectively bind to the $\beta_2$-adrenergic site and has been considered inert for more than 30 years [6]. Only after the clinical acceptance and use of racemic albuterol was the technology to separate stereoisomers (enantiomers) developed, enabling the investigation of the individual properties of (R)- and (S)-albuterol. Since then, experiments with animal, tissue, and cell culture models have suggested that (S)-albuterol has proinflammatory effects [7,8] and that it enhances airway tissue hyperresponsiveness and contractile responses [9-11]. Furthermore, (S)-albuterol has a 10-fold slower rate of metabolism than (R)-albuterol and, with frequent dosing, can accumulate in patients’ plasma and lung tissue in the absence of (R)-albuterol [12-14]. A more recent report has corroborated the proinflammatory dose- and time-dependent effects of (S)-albuterol and has discussed some of the mechanisms and pathways involved [15].

The development of new technology allowing the separation of the (R)- and (S)-albuterol stereoisomers has resulted in the development of a pure levalbuterol formulation (Xopenex, Sepracor, Marlborough, Mass). This formulation has been approved for the treatment of bronchoconstriction in patients 6 years and older, but justification of its higher acquisition cost relative to its therapeutic efficacy is controversial. The purpose of this study was to compare the overall disposition and economic impact on patients with acute asthma treated in the ED setting who received either levalbuterol or racemic albuterol as part of their primary therapy.

2. Materials and methods

All consecutive cases of patients presenting to the ED with acute asthma were retrospectively reviewed for 9 months (June 2000 through February 2001) after the addition of levalbuterol to the formulary at Muhlenberg Regional Medical Center in Plainfield, NJ. Patients 1 year or older with a primary or secondary diagnosis of acute asthma who required nebulization with a short-acting $\beta_2$-agonist, (R, S)-albuterol (racemic albuterol) or (R)-albuterol (levalbuterol), were included. Patients younger than 1 year were excluded because of the potential for a diagnosis of bronchiolitis. Patients also were excluded if they did not receive $\beta_2$-agonist treatment in the ED. Patients received either racemic albuterol 2.5 mg or levalbuterol 1.25 mg delivered via nebulizer in addition to other standard treatments including corticosteroids and oxygen. All medications and treatment regimens were determined by the treating physician.

Each case was stratified by the type of $\beta_2$-agonist administered and by age. Patient disposition was assessed (admission vs discharge). Emergency department length of stay (LOS) and objective measures of patient acuity upon
arrival, including respiratory rate, peak flow, and pulse oxygenation, were compared between treatments. $\chi^2$ Test or Fisher exact test was used to determine treatment-related differences in hospital admission rates. Emergency department LOS and arrival acuity measures were compared using analysis of variance to identify statistically significant differences between the treatment groups. A pharmacoeconomic assessment was performed to quantify the economic charge–based impact of patients in both groups. All hospital charges submitted to patients were collected, assessed, and compared between albuterol and levalbuterol groups.

In an effort to reproduce the disposition results obtained at Muhlenberg Regional Medical Center, a similar retrospective chart review was performed at Mercer Hospital, Trenton, NJ. A total of 186 consecutive cases of acute asthma presenting to the ED were reviewed, with the same inclusion and exclusion criteria, from August 1, 2002, through October 31, 2002. Each case was stratified by the type of $\beta_2$-agonist administered and by age. Patient disposition was similarly assessed (admission vs discharge). No pharmacoeconomic data were obtained from Mercer Hospital.

### 3. Results

A total of 736 consecutive cases at Muhlenberg Regional Medical Center meeting inclusion and exclusion criteria were reviewed, with 608 patients receiving racemic albuterol and 128 patients receiving levalbuterol during the 9-month period. Between the 2 treatment groups, there were no significant differences by analysis of variance in patient age, sex, ED LOS, or patient acuity (respiratory rate, peak flow, pulse oxygenation) upon ED arrival (Table 1). There were 98 (13.3%) patients admitted and 638 (86.7%) patients discharged from the ED in this consecutive patient series. Hospital admission rates were significantly lower in the levalbuterol-treated patients vs racemic albuterol–treated patients (4.7% vs 15.1% respectively; $P = .0016$ by $\chi^2$; Table 2). The racemic albuterol admission rate was comparable with the 16.4% average admission rate measured at Muhlenberg Regional Medical Center in the previous 3 years. In adult patients (age $\geq 18$ years; $n = 502$), the admission rate was 4.5% in those patients treated with levalbuterol vs 17.6% admit rate for those treated with the racemate ($P = .00085$ by Fisher exact test). In pediatric patients (age $< 18$ years; $n = 234$), the admission rate was also significantly lower for those treated with levalbuterol compared with those treated with racemic albuterol (5.0% vs 9.8%; $P = .01$ by Fisher exact test; Table 2).

The mean patient hospital charges for ED treatment in each treatment group (Table 3) were $404.56 \pm 193.56$ and $422.30 \pm 230.61$ for levalbuterol and racemic albuterol, respectively, but this difference was not statistically significant ($P = .467$). A medical chart and billing review of the 98 patients who were admitted to Muhlenberg Regional Medical Center during the study period determined that the mean LOS was 3.8 days with an average per diem cost of $945 resulting in a mean hospitalization cost of $3625 per admitted patient. Muhlenberg Regional Medical Center received approximately 1000 patients in the calendar year 2000 (approximately 3.0% of total ED volume) with acute asthma. An average of 4 nebulized $\beta_2$-agonist treatments were given to each asthmatic patient in the ED during this review. Data review from the pharmacy revealed that the cost of racemic albuterol was $0.26 per 2.5-mg dose (approximately $1.00 per ED patient treatment) and the cost of levalbuterol was $1.47 per 1.25-mg dose (approximately $6.00 per ED patient treatment). The ED admission rates for levalbuterol and racemic albuterol approximated 5% and 15%, respectively. Using a per diem rate of approximately $1000 and an approximate LOS of 4 days, the average asthma hospital stay costs approximately $4000. When one considers 1000 ED asthmatic patients presenting to the ED annually, the overall drug cost per 1000 albuterol-treated patients

### Table 1 Patient characteristics and ED statistics

<table>
<thead>
<tr>
<th>Age</th>
<th>Rac Alb admitted</th>
<th>Rac Alb discharged</th>
<th>Lev admitted</th>
<th>Lev discharged</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt;18 y</td>
<td>6.7 ± 4.9</td>
<td>6.8 ± 4.8</td>
<td>7.4 ± 5.0</td>
<td>7.2 ± 5.0</td>
<td>.88</td>
</tr>
<tr>
<td>Age ≥18 y</td>
<td>49.8 ± 17.0</td>
<td>49.2 ± 19.0</td>
<td>40.2 ± 14.1</td>
<td>39.9 ± 13.4</td>
<td>.957</td>
</tr>
<tr>
<td>Male (%)</td>
<td>48.4</td>
<td>33.3</td>
<td>50.0</td>
<td>41.0</td>
<td>.668</td>
</tr>
<tr>
<td>LOS (min)</td>
<td>331 ± 195</td>
<td>335 ± 212</td>
<td>181 ± 113</td>
<td>196 ± 107</td>
<td>.762</td>
</tr>
<tr>
<td>RR$_0$</td>
<td>29.8 ± 8.1</td>
<td>39.8 ± 14.2</td>
<td>25.6 ± 8.5</td>
<td>30.2 ± 10.3</td>
<td>.143</td>
</tr>
<tr>
<td>PF$_0$</td>
<td>200 ± 70</td>
<td>200 ± 80</td>
<td>265 ± 102</td>
<td>182 ± 70</td>
<td>.433</td>
</tr>
<tr>
<td>SAT$_0$ (%)</td>
<td>92.8 ± 10.9</td>
<td>94.0 ± 3.5</td>
<td>97.3 ± 2.4</td>
<td>96.7 ± 3.2</td>
<td>.407</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD unless noted otherwise. Rac Alb indicates racemic albuterol; Lev, levalbuterol; RR$_0$, respiratory rate; PF$_0$, peak flow; SAT$_0$, oxygenation saturation.

### Table 2 Muhlenberg Regional Medical Center patient disposition results: levalbuterol vs racemic albuterol

<table>
<thead>
<tr>
<th></th>
<th>Lev admitted</th>
<th>Rac Alb</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients (N = 736)</td>
<td>128</td>
<td>608</td>
<td></td>
</tr>
<tr>
<td>Admission to hospital from ED (age &gt; 18 y) (n = 502)</td>
<td>6 (4.7)</td>
<td>92 (15.1)</td>
<td>.0016</td>
</tr>
<tr>
<td>Admission to hospital from ED (age &lt; 18 y) (n = 234)</td>
<td>4 (4.5)</td>
<td>73 (17.6)</td>
<td>.00085</td>
</tr>
<tr>
<td>Children (age &lt; 18 y) (n = 234)</td>
<td>194</td>
<td>19 (9.8)</td>
<td>.01</td>
</tr>
</tbody>
</table>
is approximately $1000 ($1.00 per patient treatment × 1000 patients). If levalbuterol is substituted, the overall drug cost per 1000 ED asthmatic patients is $6000 ($6.00 per patient treatment × 1000 patients). Thus, an economic investment of $5000 occurs annually for the use of levalbuterol in all ED patients with acute asthma. However, the return on this investment is clearly demonstrated by examining the admission costs of care. It is projected that approximately 150 patients will be admitted for each 1000 patients treated with racemic albuterol (1000 patients × 0.15 admit rate) vs 50 admissions (1000 patients × 0.05 admit rate) for those patients treated with levalbuterol. Thus, the total costs for admission in the racemic albuterol group is $600 000 (150 admitted patients × $4000 total hospital stay costs for 4 day LOS) vs $200 000 (50 admitted patients × $4000) for patients treated with levalbuterol. The inpatient cost savings of $400 000 resulting from an investment of $5000 cost for levalbuterol results in an 80:1 risk-benefit ratio favoring levalbuterol.

The results from Mercer Hospital also revealed significant decreases in hospital admission rates with levalbuterol in comparison with the racemic drug (13.8% vs 28.9%; \( P = .021 \) by \( \chi^2 \); Table 4). Although not powered for appropriate sample sizing when stratified by age, trends toward statistical significance were noted in both adult and pediatric age groups.

### 4. Discussion

Drug therapy with isomeric derivatives of racemic compounds is not a new concept. Many single-isomer drugs are now available for clinical use such as levofloxacin, simvastatin, and enalapril. Although the (R)- and (S)-albuterol isomers have the same molecular weight and other similar physiochemical properties, they are nonsuperimposable images with regard to their 3-dimensional structure. This conformational stereochemistry confers different and distinctive properties to each isomer such that they are considered different compounds, and thus, regulatory authorities have mandated that the potential risks associated with racemic mixtures should be quantified [16]. To this end, levalbuterol was developed to maximize therapeutic effects and potentially minimize untoward effects that may be related to (S)-albuterol.

The findings shown in this study demonstrate that the use of levalbuterol when compared against racemic albuterol for the treatment of patients presenting to the ED with acute asthma significantly reduces hospital admission rates (4.7% vs 15.1%; \( P = .0016 \)) and suggest that levalbuterol has clinical advantages over racemic albuterol in critical care settings. This clinical benefit was evident in both the pediatric and adult asthmatic patients who had been treated with levalbuterol. Similar observations of the data collected from Muhlenberg Regional Medical Center, performed on consecutive patients (\( N = 186 \)) presenting with acute asthma to the Mercer Hospital ED, also found significant decreases in hospital admission rates with levalbuterol in comparison with the racemic drug. Thus, in geographically distinct ED settings, with varying physicians, patient populations, and socioeconomic conditions, the substitution of racemic albuterol with levalbuterol improved patient outcomes by reducing hospital admission rates. The observational pooled data include 922 patients with composite admission rate of 17.4% for patients treated with racemic albuterol and 7.8% for those patients treated with levalbuterol (\( P = .001; \chi^2 \)). The comparison of admission rates by site of study is shown in Fig. 2.

Previous ED studies have suggested that levalbuterol produces greater bronchodilation than racemic albuterol and improves health resource use and discharge rates [17-21]. Compared with racemic albuterol 2.5 mg, levalbuterol 1.25 mg significantly improved forced expiratory volume in 1 second percent change from baseline in adults with acute asthma (39% vs 74%, respectively; \( P < .05 \)), although levalbuterol 0.63 mg was no different from 2.5 mg of the
r Racemate (37% vs 39%, respectively) [19]. Furthermore, a post hoc analysis found that patients’ baseline (S)-albuterol plasma levels negatively impacted patients’ baseline forced expiratory volume in 1 second as well as pulmonary function 1 hour after ED treatment was begun [19]. In another randomized double-blind study, Carl et al [18] compared treatment with levalbuterol 1.25 mg with racemic albuterol 2.5 mg in more than 500 pediatric patients reporting to the ED of a major children’s hospital. Patients receiving levalbuterol had significantly reduced hospital admission rates in comparison with those receiving racemic albuterol (36% vs 45%; \( P = .021 \)). However, there was no significant difference between treatment groups for number of nebulized treatments received in the ED or ED LOS, and upon admission to the hospital, the LOS in the hospital’s asthma care unit was not significantly different between treatment groups.

Pikarsky and Acevedo [20] recently reported data from a levalbuterol hospital conversion where racemic albuterol 2.5 mg every 4 hours was replaced with levalbuterol 0.63 mg every 6 hours or levalbuterol 1.25 mg every 8 hours. Despite that levalbuterol treatments were less frequent, these patients required fewer rescue treatments for breakthrough symptoms than did those treated with racemic albuterol (with or without concomitant ipratropium bromide). Furthermore, less frequently scheduled treatments reduced workload demands on respiratory therapists and reduced the number of missed treatments because of unavailability of the therapist [21].

Questions have been raised over the cost of levalbuterol and racemic albuterol, which has generic formulations [22]. Data presented here indicate that levalbuterol is effective for the treatment of acute asthma in the ED and that, despite the higher drug cost of levalbuterol, when total system costs are considered, overall ED treatment costs were similar (5% decrease in cost in the levalbuterol group vs racemic albuterol group was not significant; \( P = .467 \)). When considering the expenses related to asthma treatment, our findings are consistent with other reports that examine parameters besides drug cost alone [21,23–25]. Truitt et al [23] reported that levalbuterol-treated inpatients with acute asthma or chronic obstructive pulmonary disease required significantly fewer \( \beta_2 \)-agonist treatments and a 67% decrease in readmissions within 30 days of discharge compared with racemic albuterol (\( P = .056 \)). Hospital use of levalbuterol translated into a nearly 1-day shorter hospital stay (\( P = .015 \)) and an average cost savings of $556 per levalbuterol-treated patient (\( P = .013 \)) [23]. In a case-controlled assessment of claims data, levalbuterol was associated with a $435 reduction in mean charges compared with a $311 increase for albuterol in outpatients receiving 2 controller medications or more [26]. Additional cost reductions associated with levalbuterol were noted with increasing asthma severity [26]. Other studies comparing levalbuterol with racemic albuterol as the primary short-acting \( \beta_2 \)-agonist therapy have reported decreased numbers of daily treatments, fewer as-needed treatments, and reductions in staffing requirements [21,25]. Therefore, in addition to cost savings associated with less frequent dosing of levalbuterol compared with racemic albuterol [27,28], levalbuterol appears to be cost-effective in the treatment of acute asthma in the ED, despite its higher drug cost. Data presented here demonstrate that the overall ED treatment costs were similar in the levalbuterol vs racemic albuterol group (\( P = .467 \)); however, significant cost savings were attained by reducing hospital admissions.

The number needed-to-treat (NNT) required to achieve clinical benefit using levalbuterol in this retrospective review is 9.6 patients (NNT = 1/0.151 – 0.047). Thus, approximately 10 patients would need to be treated with levalbuterol instead of racemic albuterol to achieve one more discharge than expected, and for every $50 ($5.00 per patient in increased levalbuterol costs) invested in treating 10 patients with levalbuterol, $4000 is saved from a resulting extra discharge. The NNT reported in this study is similar to that of other investigators [18,29] and was similarly observed in the repeat study of an additional 186 consecutive ED patients with acute asthma reported by Schreck et al [30] (Table 4).

It is very interesting that the clinical and preclinical benefits of levalbuterol compared with racemic albuterol reported in the literature cited in this investigation have occurred when the only difference between the 2 agents is the presence of the \( S \)-isomer in the racemic mixture. This suggests that the \( S \)-isomer may not be inert and instead may have some proinflammatory effect.

Limitations of this study include its retrospective design, lack of standardization in treatment regimens, smaller
numbers of patients in the levalbuterol group relative to the racemic albuterol group, and lack of postdosing efficacy comparisons regarding parameters such as peak expiratory flow rates and respiratory rates. Because of the retrospective design, no cause and effect conclusions can be drawn from the data. In addition, other treatments were not considered in our evaluation of the patient disposition of each treatment group including corticosteroids and ipratropium bromide. It should also be stated that the diagnoses of asthma for patients in this investigation are based on the reported physician diagnosis as listed in the medical record as noted on chart review. It is conceivable that some older patients, although a small portion of the sample size (11% of the racemic albuterol group), may have actually had chronic obstructive pulmonary disease. However, the large size of this observational study has hopefully allowed any treatment selection bias to be randomly distributed between the 2 study groups.

5. Conclusion

Our findings suggest that levalbuterol, when used in place of racemic albuterol, is cost-effective and reduces the number of hospital admissions in the treatment of acute asthma in the ED setting. This observation is provocative because the only difference between the 2 agents is that of the presence of the S-isomer in the racemic mixture. A prospective trial is warranted to further validate these findings.

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Defining the outcomes of risk stratification studies of ED patients with chest pain: the marginal value of adding revascularization to the composite end point

Esther H. Chen MD*, Frank Sites RN, MHA, Frances S. Shofer PhD, Judd E. Hollander MD

Abstract

Objectives: Cardiovascular risk stratification studies use various end points, sometimes including revascularization. We assessed whether adding revascularization to a strictly defined composite end point of death, acute myocardial infarction (AMI), and unstable angina (UA) impacts the likelihood of patients attaining the composite end point.

Methods: We conducted a secondary analysis of a prospectively collected data set of emergency department patients who received an electrocardiogram for chest pain. Patients were followed daily; discharged patients had 30-day telephone follow-up. The main outcome was a 30-day composite end point of death, AMI, and UA compared with death, AMI, UA, and revascularization.

Results: There were 4492 patients enrolled (mean age, 52 ± 16 years; men, 41%; African American, 68%). One hundred seventy patients were revascularized (158 had AMI or UA). Overall, the incidence of death/AMI/UA was 20.1% (95% confidence interval, 18.9%-21.2%). With revascularization included, the incidence of the composite end point was 20.3% (95% confidence interval, 19.1%-21.5%).

Conclusion: When both AMI and UA are strictly defined, there appears to be a limited role for adding revascularization to a composite end point of death, AMI, and UA because most revascularized patients have a diagnosis of AMI or UA.

1. Introduction

Patients with acute chest pain represent about 5.3% of all emergency department (ED) visits [1] and challenge the emergency physician (EP) to identify, treat, and prevent adverse events in high-risk patients, as well as to reduce unnecessary hospital admissions in low-risk patients. In response to emerging clinical trials and epidemiological studies, it became necessary to standardize outcome measures. Management guidelines for acute coronary syndromes (ACSs) were recently updated in attempts to incorporate a wide range of studies into a comprehensive evidence-based approach [2,3]. However, interpretation of results is difficult because inclusion criteria, historical parameters, and outcome measures vary among studies [4]. Subsequently, the Emergency Medicine Cardiac Research and Education Group initiated an effort to standardize operational definitions and reporting criteria to facilitate
cross-study comparisons. These proposed criteria have recently been endorsed by the Society of Academic Emergency Medicine, American College of Emergency Physicians, American Heart Association, and American College of Cardiology [5].

After these standardization efforts, it is useful to evaluate the importance of various clinical end points used in ACS studies. Risk stratification studies of ED patients with chest pain have used death, acute myocardial infarction (AMI), and unstable angina (UA) as clinical outcome measures, whereas others also include revascularization in the composite end point. In the standardized reporting guidelines for studies evaluating risk stratification of ED patients with potential ACSs [5], revascularization was not considered a mandatory criterion to be reported. Therefore, we questioned whether the absence of this criterion would impact the results of these studies. We hypothesized that the addition of revascularization to a composite end point of death, AMI, and UA would not alter the likelihood of achieving a negative composite end point.

2. Materials and methods

2.1. Study design

This was a secondary analysis of a prospectively collected data set of ED patients with chest pain. We assessed whether adding revascularization to a strictly defined composite end point of death, AMI, and UA impacts the likelihood of patients attaining the composite end point. The study was approved by the University of Pennsylvania Committee on Research Involving Human Subjects.

2.2. Study setting and population

Patients were enrolled at an urban tertiary care university hospital ED with an annual census of approximately 51,000 visits during the study period, from July 1999 to March 2002. Patients were included if they were older than 24 years with chest pain prompting an electrocardiogram (ECG) and younger than 24 years only if they used cocaine. Broad inclusion criteria were intentionally chosen to ensure generalizability of the data.

2.3. Study protocol and measurements

Trained research assistants enrolled ED patients 16 hours per day, 7 days per week, which captures 85% to 95% of eligible patients. Information collected for each patient included demographics, historical description of chest pain characteristics, laboratory data, and ECG data. Hospitalized patients were followed daily for complications and interventions. Clinical information was obtained from the treating physician. Determination of final diagnosis and cardiac complications was made during daily communication between the investigators and the healthcare team. Postdischarge medical record review was not used. A 30-day follow-up for all study subjects was obtained by a standardized telephone interview.

2.3.1. Cardiac biomarker assays

Venous blood samples were collected on presentation to the ED in phlebotomy tubes containing no anticoagulant or preservative. Cardiac troponin I and creatine kinase–MB (CK-MB) were measured by an enzyme-linked immunosorbent assay using an Abbott AxSYM automated analyzer (Abbott Laboratories, Mountain View, Calif).

2.3.2. Main outcome

The main outcome measure was the 30-day composite end point of death, AMI, and UA compared with the composite end point of death, AMI, UA, and revascularization.

Death was defined as all-cause mortality. AMI was defined in accordance with the European Society of Cardiology/American College of Cardiology 2000 consensus definition [6]. A diagnosis of AMI was made if the patient had an elevation of cardiac troponin I of 2 ng/mL or greater or CK-MB enzyme of 10 ng/mL or greater. UA was defined using standardized criteria [5,7]. UA was considered to occur if there was a documented reversible ischemia on stress test, coronary artery occlusion of 70% or higher in at least 1 vessel as seen during cardiac catheterization, or elevations of cardiac enzymes greater than laboratory normal but less than levels necessary for diagnosis of AMI (troponin I, ≥0.4 ng/mL but <2 ng/mL; CK-MB, ≥5 ng/mL but <10 ng/mL) [7]. We did not use the Canadian Cardiovascular Society or Agency for Health Care Policy and Research Clinical Practice Guideline descriptions of UA because they assume a diagnosis of ischemic chest pain and do not apply to unselected ED patients with chest pain. Revascularization was defined as percutaneous coronary intervention (PCI) and coronary artery bypass graft (CABG).

2.4. Data analysis

For patients with multiple visits, presentation characteristics and outcomes were counted separately for each visit, but age, race, and sex were counted only once. Outcome data are presented with percentage of frequency of occurrence, with 95% confidence intervals (CIs) provided for main outcomes. Data were analyzed using SAS statistical software (Version 8.2; SAS Institute, Cary, NC).

3. Results

There were 3819 patients who presented to the ED 4492 times during the study. Patients had a mean age of 51.8 ± 17.4 years and were more likely to be women (59%) and African American (68%). Table 1 shows their demographic and historical characteristics. Of the total cohort, 1674 patients were discharged to home (37%); the remainder was admitted to the hospital. There were 2147 patients admitted to telemetry (48%); 496 admitted to the intensive care unit (11%); 135 admitted to unmonitored
floor beds (3%); 14 admitted directly to the catheterization laboratory without another inpatient bed assignment (<1%); 13 patients signed out against medical advice, and 10 patients were transferred to another hospital. Two patients died before admission and 1 patient left before a complete evaluation could be performed. Thirty-day follow-up information was obtained for 98% of the study patients. During the hospitalization, 319 patients sustained an AMI (7%) and 545 patients were diagnosed with UA (12%). In addition, 29 patients died, 11 developed a late myocardial infarction, 128 had PCI, and 26 received CABG (4 patients received PCI before CABG). At 30-day follow-up, 20 patients sustained an AMI, 17 patients had PCI, 11 patients received CABG, and 54 patients died. Eight of the revascularized patients also had PCI during the index hospitalization.

With respect to the main outcome, 170 patients had revascularization performed. Of these, 158 had a diagnosis of AMI or UA during initial hospitalization. Overall, the incidence of the triple composite end point was 20.1% (95% CI, 18.9%-21.2%). When revascularization was added to this composite outcome, the incidence of the composite end point was 20.3% (95% CI, 19.1%-21.5%). Thus, the addition of revascularization to the triple composite outcome did not alter the overall likelihood of reaching the composite outcome.

4. Discussion

EPs are unable to accurately identify patients at very low risk for ACS. More than 2% of patients with ACS are sent home from the ED, with a higher 30-day mortality compared with hospitalized patients [8]. These patients are more likely to present with atypical features and have normal or nondiagnostic ECGs. To reduce the number of missed diagnoses and standardize emergency care of patients with chest pain, practice guidelines that incorporate risk stratification tools and current therapies were developed [2,3]. Unfortunately, these expert recommendations have not been uniformly adhered to, and these risk stratification tools have not been widely accepted.

Several solutions have been proposed to solve the problem of noncompliance with expert guidelines and risk stratification algorithms. For example, the CRUSADE initiative takes a multidisciplinary approach to improving the care of patients with ACS by encouraging adherence to guidelines, implementing ED-focused educational interventions, and providing a national registry of high-risk patients with chest pain, a tool for continuous quality improvement [9]. In addition, Emergency Medicine Cardiac Research and Education Group, an international collaboration of emergency health-care professionals, has taken a multifaceted approach to improving the diagnosis and treatment of ACS by involving EPs in establishing clinical pathways, encouraging collaborations with cardiologists in clinical trials, and standardizing reporting criteria in cardiovascular research. Standardizing reporting criteria of operational and outcome definitions enables EPs to perform more rigorous scientific research and to compare clinical outcomes of patients in trials that use various risk stratification tools [5].

This study specifically addressed whether adding revascularization to a strictly defined triple composite end point of death, AMI, and UA will alter the incidence of patients who reach a composite outcome. We found that the likelihood of attaining the outcome was the same with or without the use of revascularization in the composite index. The most obvious explanation for this finding is that most patients who received revascularization had a diagnosis of AMI or UA during their hospital visit. In fact, this was true for 158 (93%) of the 170 patients who received revascularization.

5. Limitations

Our study population was predominantly women and African American. Therefore, our results may not generalize to other patient populations. Furthermore, because this was a single institution study, differences in revascularization rates (local, regional, sex, and ethnic) could not be assessed and may further limit the generalizability of this study.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. of study patients (n = 4492 visits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>51.8 ± 15.9</td>
</tr>
<tr>
<td>Ethnicitya</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>69 (2)</td>
</tr>
<tr>
<td>Black</td>
<td>2613 (68)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>43 (1)</td>
</tr>
<tr>
<td>Other/unknown</td>
<td>31 (1)</td>
</tr>
<tr>
<td>White</td>
<td>1063 (28)</td>
</tr>
<tr>
<td>Sexa</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1550 (41)</td>
</tr>
<tr>
<td>Female</td>
<td>2269 (59)</td>
</tr>
<tr>
<td>Cardiac risk factors</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>2195 (49)</td>
</tr>
<tr>
<td>Family history of premature CAD</td>
<td>903 (20)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>806 (18)</td>
</tr>
<tr>
<td>Tobacco use</td>
<td>1685 (38)</td>
</tr>
<tr>
<td>Hypercholesterolemia/hyperlipidemia</td>
<td>789 (18)</td>
</tr>
<tr>
<td>Cocaine use</td>
<td>104 (2)</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>497 (12)</td>
</tr>
<tr>
<td>Known CAD</td>
<td>878 (20)</td>
</tr>
<tr>
<td>Overall ECG impression</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>1912 (43)</td>
</tr>
<tr>
<td>Early repolarization only</td>
<td>171 (4)</td>
</tr>
<tr>
<td>Nonspecific/nondiagnostic changes</td>
<td>2085 (50)</td>
</tr>
<tr>
<td>Ischemia</td>
<td>224 (5)</td>
</tr>
<tr>
<td>AMI</td>
<td>100 (2)</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD or n (%). CAD indicates coronary artery disease.

a Age, sex, and ethnicity are only counted once per patient (n = 3819) regardless of number of visits.
Although we used standard definitions for the adverse outcomes, we were unable to standardize the interventional strategies for our patients and relied upon the clinical judgment of our cardiologists.

We attempted to reduce the limitations that are pervasive in studies of ED patients with chest pain. Trained research assistants enrolled patients prospectively into the study to reduce selection bias. Standardized definitions of adverse outcomes were used and strictly enforced. Inhospital tracking was performed daily rather than relying on medical record review, which reduced misclassification bias.

6. Conclusions

There appears to be a limited role for adding revascularization to a triple composite end point of death, AMI, and UA when these items are strictly defined because most revascularized patients have a diagnosis of AMI or UA.

References


Original Contributions

Correlation of arterial PCO₂ and PETCO₂ in prehospital controlled ventilation

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Accepted 15 April 2005

Abstract

Introduction: This study was carried out to estimate the relationship between arterial PCO₂ (PaCO₂) and end-tidal carbon dioxide (PETCO₂) during prehospital controlled ventilation and also to evaluate variation of the gradient between PCO₂ and PETCO₂ during prehospital transport.

Methods: Measurements of PETCO₂ from capnography values and PaCO₂ from arterial blood gases were registered at the beginning (T₀) and at the end (T_end) of out-of-hospital management. For all patients requiring invasive ventilation, the gradient between PCO₂ and PETCO₂ was calculated for T₀ and T_end, the PaCO₂ – PETCO₂ variation between T_end and T₀ was also calculated.

Results: One hundred patients were included in this study (mean age, 58.4 ± 16.4 years; 57 were male). There was no variation of the mean gradient (ΔPaCO₂ – PETCO₂) during transport (8.64 ± 13.5 mm Hg at T₀ and 7.26 ± 12.94 mm Hg at T_end). Thirty-six percent of patients (n = 36) had a gradient above +10 mm Hg, and for 6% of patients (n = 4) the gradient was lower than −10 mm Hg. The PaCO₂ – PETCO₂ gradient was not significantly different according to the pathology, but was significantly higher in hypercapnic patients compared with hypocapnic or normocapnic patients. In patients with severe head injury, the capnia was normalized in 80% of patients at the end of the transport according to the last blood gas result. In this subgroup the ΔPaCO₂ – PETCO₂ (T_end – T₀) gradient was stable between T₀ and T_end except in 20% of the patients for whom the ΔPaCO₂ – PETCO₂ was lower than −10 mm Hg. Fifty-four percent of critical care physicians had modified the respiratory setting after the first arterial blood gas results.

Conclusions: The PaCO₂ cannot be estimated by the PETCO₂ in the prehospital setting. There is wide variation in the gradient between PCO₂ and PETCO₂ depending on patient condition, and over time, the relationship does not remain constant and thus cannot be useful in prehospital ventilation management. © 2005 Elsevier Inc. All rights reserved.

1. Introduction

Prehospital critical care teams manage critically ill patients. Endotracheal intubation and controlled ventilation, initiated in the field, are required in these emergency
Correlation of arterial P\textsubscript{CO}\textsubscript{2} and P\textsubscript{ETCO}\textsubscript{2} in prehospital controlled ventilation

2. Patients and methods

2.1. Study setting and population

This prehospital study has been carried out in the French emergency medical services system (SAMU) based on physician-staffed ambulances over a 16-month period. The French emergency medical services system is based on 2 types of ambulances: emergency medical technician (EMT)-staffed ambulances for basic life support and physician-staffed ambulances for advanced life support [16,17]. The EMTs are members of the fire department or members of the French Red Cross. The telephone number is a national emergency number. Switchboard operators, available 24 hours a day, receive all calls in the dispatching center (SAMU) and transmit them to the dispatching physician, who, depending on the situation, decides to send out a team of EMTs (who are members of the fire department rescue services) and, at the same time, according to the potential severity, an ambulance staffed by the medical team. Because of the greater number of EMTs, they are frequently closer to the patient and can start basic life support before the arrival of the medical team. The medical unit provides advanced life support on scene and during transportation to the hospital. The medical team physician and the dispatching physician are in contact throughout the intervention, and the dispatching center locates the hospital best suited to the patient’s critical condition. The medical team comprises a paramedic, a physician, and a third person who can be a resident, a medical student, or a nurse anesthetist. All physicians were senior physicians who had more than 2 years of experience in prehospital care, supervised and trained by anesthesiologists.

All consecutive patients, older than 18 years who required an endotracheal intubation and controlled ventilation outside hospital, were enrolled in this study.

2.2. Study protocols

This protocol was approved by an ethics committee. This was a unicoentric, prospective, and descriptive study. All patients included in this study were continuously monitored by capnometry, as is usual in our practice. 

ECO\textsubscript{2} was measured with a mainstream capnometer using infrared absorption spectroscopy [18] (capnometer module for PROPAC encore, Welch Allyn Protocol, Inc, Beaverton, Ore). For each intubated patient, a carbon dioxide airway adapter (Welch Allyn Protocol, Inc) was connected to the endotracheal tube, the airway filter, and the ventilator circuit tubing. The mainstream carbon dioxide sensor was connected to our monitoring system (PROPAC encore), which gives continuous values of PETCO\textsubscript{2} with waveforms. This system was calibrated and controlled regularly by a registered company (at least every year).

To evaluate the agreement between PETCO\textsubscript{2} values and PaCO\textsubscript{2} values, arterial sampling was performed. Two arterial blood gas samples were taken during the critical care intervention by the medical team: at the beginning of the intervention ([T\textsubscript{0}]) and at the end (time end [T\textsubscript{end}]) on arrival at the hospital. The first arterial blood gases were done after a stabilizing ventilation period (around 15 minutes) with transport ventilators (OXYOLOG 2000 Company or T-BIRD, Sebbac). The arterial blood gases were analyzed by a portable blood gas analyzer (I-STAT, I-STAT Corporation, East Windsor, NJ). This portable blood gas analyzer was calibrated every morning by our nurse anesthetist or paramedic and controlled by a registered company at least every 6 months. The correlation between blood gases results from the I-STAT analyzer and blood gases results from the standard hospital analysis have been validated in the literature [18-20].

Ventilation settings on the portable ventilators were defined by the physician on board the ambulance according to the national recommendations [2] and to the patient’s weight and condition. Ventilation settings were then adjusted according to the first arterial blood gas results. In addition, anesthetic drugs were left to the individual physician’s discretion in accordance with national guidelines regarding anesthesia in the field, most often etomidate.
and suxamethonium [21-23]. The sedative-analgesia protocol to adapt the patient to his/her ventilation was also left to the individual physician’s choice according to the national recommendations, which are most often midazolam and fentanyl [2,24].

2.3. Methodological limitations

This study is a descriptive study based on our observations. The sample size was arbitrarily chosen because the aim of this study was to focus on our observed values. Because this study’s aim is not to highlight a statistical difference between 2 groups, the sample was not calculated according a primary end point. Thus, 100 patients are sufficient to make correct statistical analysis.

2.4. Measurements

The hemodynamic (heart rate and blood pressure) and respiratory parameters (oxygen saturation and respiratory rate) were continuously monitored in the field and during transportation and recorded by the medical team. Medical background (such as asthma or BPCO), notion of severe head injury, suspected diagnosis and ventilation setting, and anesthetic or analgesic treatments were also registered. Arterial blood gas samples were done by the physician or by the nurse anesthetist at $T_0$ and $T_{end}$ and were analyzed by the physician. Hypercapnia was defined as $P_{aco_2}$ of 43 mm Hg or higher, and hypocapnia was defined as $P_{aco_2}$ of 34 mm Hg or lower after arterial blood gas results. For each patient the $P_{aco_2} - P_{etco_2}$ gradient at $T_0$ and $T_{end}$ was calculated. To appreciate the time variation of the $P_{aco_2} - P_{etco_2}$ gradient, the gradient $\Delta P_{aco_2} - P_{etco_2}$ ($\Delta P_{aco_2} - P_{etco_2} = T_{end} - T_0$) was also calculated.

Any modifications or absence of modifications carried out by the physician on respiratory settings according to the results of the first arterial blood gases were also recorded.

2.5. Statistical analysis

Quantitative variables are expressed as mean ± SD and also as frequency distribution. Qualitative variables are expressed as frequency distribution with a 95% confidence interval. Qualitative data were analyzed by the $\chi^2$ test analysis and quantitative data were analyzed by analysis of variance, completed by the Fisher test. The sample size was arbitrarily chosen because this was a descriptive study. A $P < .05$ was considered significant. Statistical analysis was done by the statistical package StatView (SAS Institute Inc, Berkeley, Calif).

3. Results

A total of 100 patients were included in this study (mean age, 58.4 ± 16.4 years; 57 were male). Patients’ disease data are noted in Table 1. Hemodynamic and ventilation data are shown in Table 2. Arterial blood gas results and calculated $P_{aco_2} - P_{etco_2}$ are recorded in Table 3. The gradient between $P_{aco_2}$ and $P_{etco_2}$ was available for 96 patients at $T_0$ and for 82 patients at $T_{end}$. The missing data for 18 patients were due to technical difficulties to obtain blood gas samples at $T_0$ or $T_{end}$.

Even if the mean gradient was not so high, the variation of the $P_{etco_2}$-$P_{aco_2}$ gradient was wide among patients (Fig. 1A and B). $P_{etco_2} - P_{aco_2}$ gradient showed important variations between −19.7 to 75 mm Hg at $T_0$ and −11.8 to 98 mm Hg at $T_{end}$. As shown in Fig. 1A and B, at $T_0$ for 36% of the patients (n = 35) and at $T_{end}$ for 27% of the patients (n = 22), the $P_{aco_2} - P_{etco_2}$ gradient’s value exceeded +10 mm Hg. At $T_0$, for 6% of the patients (n = 6), and at $T_{end}$, for 2% (n = 2) of the patients, the $P_{aco_2} - P_{etco_2}$ gradient’s value was lower than −10 mm Hg.

Table 1 Main disorders (n = 100)

<table>
<thead>
<tr>
<th>Main disorders</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurology</td>
<td>30 (30)</td>
</tr>
<tr>
<td>Cardiology</td>
<td>20 (20)</td>
</tr>
<tr>
<td>Infection</td>
<td>10 (10)</td>
</tr>
<tr>
<td>Respiratory</td>
<td>8 (8)</td>
</tr>
<tr>
<td>Traumatology</td>
<td>8 (8)</td>
</tr>
<tr>
<td>Digestive</td>
<td>3 (3)</td>
</tr>
<tr>
<td>Others</td>
<td>21 (21)</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD (range).

Table 2 Hemodynamic and ventilation variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>$T_0$</th>
<th>$T_{end}$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>121.8 ± 25.7 (70-220)</td>
<td>114.5 ± 26.4 (6-200)</td>
<td>.03</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>68.2 ± 17.0 (30-117)</td>
<td>66 ± 16.5 (17-109)</td>
<td>.03</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>92.3 ± 22.4 (35-162)</td>
<td>97.9 ± 22.6 (45-173)</td>
<td>.01</td>
</tr>
<tr>
<td>Minute ventilation (L/min)</td>
<td>9.3 ± 2.8 (2.5-17)</td>
<td>9.5 ± 3 (3-21)</td>
<td>NS</td>
</tr>
<tr>
<td>Respiratory rate (cpm)</td>
<td>15.2 ± 3.1 (8-3)</td>
<td>15.4 ± 3 (8-23)</td>
<td>NS</td>
</tr>
<tr>
<td>Fraction of inspired oxygen (%)</td>
<td>78.2 ± 20 (40-100)</td>
<td>72 ± 19 (40-100)</td>
<td>.0001</td>
</tr>
<tr>
<td>Peak pressure (mm Hg)</td>
<td>30.1 ± 9.6 (0.4-50)</td>
<td>27.9 ± 8.2 (0.4-50)</td>
<td>NS</td>
</tr>
<tr>
<td>No. of activated alarms</td>
<td>22</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>SpO$_2$ (%)</td>
<td>98.4 ± 6.0 (45-100)</td>
<td>98.7 ± 4.2 (65-100)</td>
<td>NS</td>
</tr>
<tr>
<td>Total time of ventilation</td>
<td>92.2 ± 46.9 (15-270)</td>
<td>92.2 ± 46.9 (15-270)</td>
<td>NS</td>
</tr>
</tbody>
</table>
The PaCO₂ - PETCO₂ gradient at $T_0$ was not significantly different according to the pathology but was significantly more important in hypercapnic patients ($n = 60$) than in normocapnic ($n = 21$) or hypocapnic patients ($n = 15$) (Table 4). For 4 patients, the data were incomplete: for 2 patients, ETCO₂ values were missing and for the 2 other patients, arterial blood gases were impossible to take. Indeed, the mean gradient at $T_0$ for hypercapnic patients was significantly higher (13.3 ± 14.9 mm Hg) compared with hypocapnic (1 ± 6.3 mm Hg) or normocapnic (2.6 ± 7.2 mm Hg) patients with $P = .0002$ (Table 4). The mean PaCO₂ - PETCO₂ gradient was not significantly different between $T_0$ and $T_{end}$ (9 ± 13.6 mm Hg at $T_0$ vs 7.3 ± 13 mm Hg at $T_{end}$) (Table 3), but 22% ($n = 18$) of patients have the $\Delta$PaCO₂ - PETCO₂ ($T_{end} - T_0$) gradient lower than −5 mm Hg and 18% ($n = 15$) had higher than +5 mm Hg (Table 4).

### Table 3  Arterial blood gases results and PaCO₂ – PETCO₂ gradient (mm Hg)

<table>
<thead>
<tr>
<th>Variables</th>
<th>$T_0$</th>
<th>$T_{end}$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pH (mm Hg)</td>
<td>7.30 ± 0.2 (6.7 to 7.6)</td>
<td>7.34 ± 0.2 (6.7 to 7.6)</td>
<td>.0003</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>48.4 ± 17.7 (21-130)</td>
<td>44.1 ± 14.6 (23.2-122)</td>
<td>.0001</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>253.3 ± 152.2 (25-635)</td>
<td>240.6 ± 155.7 (28-800)</td>
<td>NS</td>
</tr>
<tr>
<td>ETCO₂ (mm Hg)</td>
<td>40 ± 13.3 (17-80)</td>
<td>36.4 ± 8.9 (23-71)</td>
<td>.0002</td>
</tr>
<tr>
<td>PaCO₂ – PETCO₂ gradient (mm Hg) calculated values</td>
<td>9.0 ± 13.6 (−19.7 to 75)</td>
<td>7.3 ± 12.9 (−11.8 to 98)</td>
<td>NS</td>
</tr>
<tr>
<td>$\Delta$PaCO₂ – PETCO₂ ($T_{end} - T_0$) calculated values</td>
<td>−1.0 ± 9.1 (−31 to 23)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD (range).

**Fig. 1**  A, PaCO₂ – PETCO₂ gradient (mm Hg) at the beginning of prehospital management ($T_0$). B, PaCO₂ – PETCO₂ gradient (mm Hg) at the end of prehospital management ($T_{end}$).
4. Discussion

The role of capnography in prehospital management is indisputable [25-27]. Thus, capnometry’s role is not any more to show in several applications: ensure correct placement of endotracheal tube [28,29], verify the integrity of mechanical ventilation equipment [26,28], and predicting outcome in patient survival. Moreover, because PETCO2 seems to be strongly linked to the cardiac output [14], PETCO2 is now used also during cardiopulmonary resuscitation to optimize chest massage [8,14,30,31]. For all these reasons, capnography is, in France, firmly recommended during intrahospital and intrahospital transport of patients [2]. The aim of this recommendation is to have a secure and a reliable monitoring of patient’s ventilation and circulation.

This study was conducted to analyze the PaCO2 – PETCO2 gradient during out-of-hospital emergency medicine, to improve the quality of ventilation in the field. According to other studies, we observed that, most patients experienced hyper- or hypocapnia during ventilation in the field. In addition, a number of patients had wide variations of the PaCO2 – PETCO2 gradient during transport. If the benefit of capnography is not being questioned in prehospital care, these results suggest that, capnography results must be carefully interpreted in patients who need a strict control of capnia.

Patients in out-of-hospital medicine are often critically ill or injured. Ventilatory and hemodynamic patient conditions are precarious and require fast endotracheal intubation and controlled ventilation. Prehospital urgent endotracheal intubation is not easy to undertake and requires fast management [17,21,22]. Various studies have demonstrated that mechanical ventilation initiated before the arrival at the hospital significantly improved the outcome for patients with severe head injuries [15,21]. However, recent studies reveal that the quality of ventilation initiated in the field is uncertain [25]. One such study, in Germany, shows that only 42.6% of patients with severe head injuries had normoventilation, whereas 40.9% had hyperventilation and 16.4% hypoventilation [32]. A French study observed that only 19% of patients with severe head injuries had normocapnia on arrival [12]. In another study, 43% of patients with severe head injuries had normocapnia upon arrival at the hospital [15]. The other patients experienced hypocapnia or hypercapnia, which carries potential risks for cerebral ischemia in this type of traumatic pathology [12,13,15]. Authors suggest that patient’s weight for the ventilator’s setting was undervalued. In another study, an incorrect adjustment of respiratory settings on the ventilator in the event of a patient’s new or previous pulmonary condition may contribute to hypo- or hypercapnia [35]. All these studies show that adaptation of out-of-hospital controlled ventilation is delicate and requires strict monitoring of ventilatory settings throughout transportation. Capnography has been proposed for ventilation as a noninvasive ventilation monitoring method, which seems to be very useful in out-of-hospital medicine. Some studies confirmed that capnography facilitates tight control of ventilation during intra-hospital [33] and prehospital transportation for intubated patients [34]. Indeed, Helm et al observed that 63% of patients with capnography monitoring had a normocapnia vs 20% of patients without capnography monitoring. In addition, the incidence of hyperventilation was reduced to 5% vs 37% without capnography monitoring [34]. A French study has shown that, even if a significant gradient exists between PETCO2 and PaCO2, monitoring of PETCO2 could be useful to avoid hypocapnia or hypercapnia in patients with severe head injuries [35].

Indeed, a physiological gradient does exist between arterial PCO2 and alveolar PCO2, reflecting the existence of physiological pulmonary dead space [36,37]. Thus, when this physiological dead space increases, especially in obstructive pulmonary pathology, hypovolemia, or mechanical ventilation, the PaCO2 – PETCO2 gradient increases [29]. This gradient has been reported to be 2 mm Hg (with a conscious patient) to 5 mm Hg (with an intubated and sedated patient) with PaCO2 exceeding PETCO2 [27,38,39]. It may be up to 20 mm Hg in patients with severe pulmonary or major systemic disease. Moreover, in a previous study, results show that mechanical ventilation, while creating some atelectasis (source of low V/Q ratio), acts on PaCO2 – PETCO2 gradient. Broadly, PaCO2 – PETCO2 variations depend on metabolic, hemodynamic, physiological, or ventilatory status, with wide variations due to life-threatening condition, frequently observed in out-of-hospital medicine.

According to these studies, because carbon dioxide’s elimination is influenced by many parameters (physiological,
pulmonary, cardiac, equipment, etc), PETCO2 cannot be an exact reflection of the PaCO2 [37,40]. Even if capnography is a useful noninvasive monitor of systemic metabolism, circulation, and ventilation, the gradient between PaCO2 and PETCO2 may vary considerably. Especially in prehospital management, where the invasive ventilation is so difficult to monitor, if it is impossible to estimate PaCO2 from the PETCO2, it could be a handicap. In our study, we observed wide variations of the PaCO2 – PETCO2 gradient between patients and during transportation so that we cannot estimate the real value of PaCO2 from the value of PETCO2. If a close control of capnia is not always necessary during prehospital care, it can be very important for several patients, such as those with severe head injuries. In these cases, removing blood gases in prehospital medicine seems to be a way to control capnia.

Many studies have shown that, especially for patients with severe traumatic head injuries, this gradient may vary so much that capnography was not a significant parameter to monitor these patients [30,42]. Russell and Graybeal [41] found a variation of the gradient in trauma patients, which was already higher than previously reported in many studies for patients during anesthesia or critical care.

Negative values of the gradient have been reported in the literature (8% of postoperative cardiac surgery patients and 2% of neurointensive care patients). Seguin et al [42] found a negative gradient for 13% of patients. Russell and Graybeal [41] also found a negative gradient for 4.1% of patients. The existence of a negative gradient is well known, but the exact mechanism has not been elucidated. It has been postulated that previously closed alveolar units slowly empty adding their higher carbon dioxide concentrations to expired breath. In others words, a lower ventilation decreases pulmonary dead space and increases the total tidal volume, which is more involved in gas exchange for areas with low V/Q, increasing ET CO2 values.

Our results are concordant with the literature. We observed a wide variation of PaCO2 – PETCO2 gradient with either very high or very low, positive and negative values [29,42]. Pathology did not influence these variations in our study. However, the higher values were found in hypercapnic patients. In addition, we observed that blood gas samples were useful for more than 50% of physicians, who modified their first ventilator settings after the first arterial blood gases results to obtain normocapnia. These results confirm that blood gas samples are useful in the prehospital setting to help physicians control the parameters of ventilation.

Time variations of the PaCO2-ETCO2 gradient has also been described in the literature, especially in prehospital care [42]. In this study, we observed that the mean ΔPaCO2 – PETCO2 (Tend – To) gradient was not so high, as observed in previous studies during anesthesia [43]. But, despite this relative time stability, about one third of patients had a time variation of their gradient of more than −5 or +5 mm Hg (Fig. 2).

These results were also observed in the study by Russell and Graybeal [41], where PETCO2 and PaCO2 varied in opposite directions for 27% of cases. Many studies since 1989 found the inability of PETCO2 changes to predict the direction of the change of PaCO2. But all these studies were intrahospital studies and the time intervals between 2 measures were shorter than in our studies [41,44]. Secondly, most of their data consisted of patients with injury or traumatic pathology. Our results suggest that, in prehospital care, capnography cannot be used for capnia monitoring for a long time if a close control of capnia is necessary. Even if the gradient has been defined at the beginning of prehospital transfer, it can be useful for some patients to control blood gas in the case of a long transfer.

Nevertheless, some limits to this study must be discussed. This was a descriptive and observational study. It

![Fig. 2](image-url)
was not a randomized study that aimed to evaluate the benefit of blood gas samples vs capnography to improve mechanical ventilation in the field. Therefore, the end point was not to demonstrate the superiority of arterial blood gases samples vs capnography alone.

In conclusion, even if the benefit of capnography is not being questioned in prehospital care, it is not a reliable means to estimate accurately the real value of PaCO2 and is not sufficient by itself to adjust respiratory setting for patients who need a close control of capnia. In addition to all the progress, which has been made in critical care equipment (ventilators [45], monitors), and alongside the advances made in the skills and knowledge of emergency physicians, arterial blood gases may also contribute to optimize the quality of prehospital mechanical ventilation. For patients who either required a tight control of PaCO2 or endured lengthy transportation, or both, arterial blood gases alongside capnography could be a useful help in prehospital management.

Acknowledgment

We thank all the physicians and nurses who took part in this study. The authors gratefully thank Mrs Fiona Richard-son for her help with the English revision and Prof Jean Mantz for his help in the response to the reviewers.

References


The prevalence of false-positive cardiac troponin I in ED patients with rhabdomyolysis

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Abstract

Objective: Cardiac troponin I (cTnI) is considered the most specific marker of cardiac muscle injury. We encountered several patients with rhabdomyolysis and elevated cTnI, although they did not otherwise have evidence of cardiac injury. We determined the prevalence of false-positive cTnI in emergency department (ED) patients with rhabdomyolysis.

Methods: We conducted a retrospective cohort study of ED patients admitted with a diagnosis of rhabdomyolysis. Patients were included in the study if they had a serum creatine kinase (CK) of 1000 U/L or greater and at least one serum cTnI determination. Patients with positive cTnI were considered true positives if they had either electrocardiography (EKG) or echocardiography abnormalities; false positives if both the EKG and the echocardiography were considered normal; or indeterminate if they did not have both an EKG and an echocardiogram. The primary outcome of the study was the prevalence of false-positive cTnI. Secondary outcomes included risk stratification by cocaine use, myoglobinuria, and renal failure and correlation of peak CK and troponin levels.

Results: One hundred nine patients were included in the final analysis; 55 (50%) patients had a positive cTnI. Of the 55 patients with positive cTnI, 32 (58%) were true positives, 18 (33%) were false positives, and 5 (9%) were indeterminate. The prevalence of false-positive cTnI was 17% (18/109, 95% confidence interval 0.10-0.25). There was no association between false-positive cTnI and cocaine use, renal failure, or myoglobinuria. There was poor correlation between peak CK and peak cTnI levels ($r = -.08$, 95% confidence interval $-0.34$ to 0.19).

Conclusion: The prevalence of false-positive cTnI in ED patients with rhabdomyolysis is 17%.

1. Introduction

Clinicians use a variety of serum markers for determining cardiac injury, such as the cardiac isoform of creatine kinase (CK), myoglobin, and cardiac troponins T and I. Of these “cardiac enzymes,” cardiac troponin I (cTnI) is considered to be the most specific for cardiac injury [1-4]. In fact, the American College of Cardiologists has adopted cTnI as the standard for serologic evidence of myocardial infarction (MI) [5]. However, we have observed several patients in our emergency department (ED) with rhabdomyolysis in whom cTnI levels were elevated, although these patients did not otherwise have evidence of myocardial injury.
reports, which has similarly reported false-positive elevations in cTnI among patients with rhabdomyolysis [6-13]. We were interested in determining the prevalence of false-positive cTnI in ED patients with rhabdomyolysis and whether cTnI levels in such patients were affected by cocaine use, renal failure, or myoglobinuria, three variables that have been associated with elevations in cTnI levels in other patient populations.

2. Methods

This was a retrospective cohort study at an urban teaching medical center. The hospital database was queried for patients admitted from the ED with rhabdomyolysis. Patients were included in the study if (1) they had a peak serum CK of 1000 U/L or greater and (2) they had at least one cTnI determination during their hospitalization. The study was approved by our Institutional Review Board.

The medical chart was reviewed to obtain demographic (age, sex) and clinical data (myoglobinuria, renal failure, urine toxicology, electrocardiography [EKG], echocardiography). Each chart was reviewed by one of the three authors and data were abstracted using a standardized data collection instrument. In our laboratory, the normal range of CK is 5 to 150 U/L and troponin less than 0.03 μg/L. A serum cTnI of 0.10 μg/L or greater is considered positive.

The troponin assay used by our laboratory is made by Beckman (Fullerton, Calif). Myoglobinuria was defined as moderate or large blood on the urinalysis dipstick with less than 10 red blood cells per high-power field on microscopy. Renal failure was defined as a serum creatinine of 2.0 mg/dL or greater. Cocaine use was determined by urine toxicology results. Electrocardiography and echocardiography results were obtained by medical record review, as interpreted by staff cardiologists. An EKG was considered abnormal if there was ischemia or evidence of acute MI as defined by the standard EKG classification system [14], bundle-branch block, or any arrhythmia excluding sinus tachycardia, regardless of the chronicity of the finding. Echocardiography was considered abnormal if there was any wall motion abnormality (segmental or global), regardless of the chronicity of the finding. A patient who did not have any of the above ancillary evaluations during their hospitalization was considered indeterminate for that particular test. A patient was considered cTnI positive if any of the cTnI determinations were positive.

Patients with positive cTnI were considered true positives if there were either EKG or echocardiography abnormalities; false positives if both the EKG and the echocardiography were considered normal; and indeterminate if they did not have both an EKG and an echocardiogram.

The primary outcome of the study was the prevalence of false-positive cTnI in patients with rhabdomyolysis. Secondary outcomes included risk stratification by cocaine use, myoglobinuria, and renal function and correlation of peak CK and peak cTnI levels. Prevalence was determined by simple proportion. Risk stratification was determined using relative risk. Correlation was analyzed using linear correlation. We estimated that 73 patients were needed to detect a 10% difference with a 5% prevalence of disease (false positives) and a 2-tailed α error of 0.05. We intended to enroll 150 patients into the study (approximately double the estimated sample size) in case the prevalence of disease was different in our patient population. By examining the hospital database, a 3-year period would provide us with approximately 150 patients, and we used this data set for our

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographics of the study population</th>
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</thead>
<tbody>
<tr>
<td><strong>Age (y)</strong></td>
<td>53 ± 21 (range, 18-94)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td>76% M, 24% F</td>
</tr>
<tr>
<td><strong>Peak CK</strong></td>
<td>14140 ± 27245 (range, 1016-156500)</td>
</tr>
<tr>
<td><strong>Peak troponin</strong></td>
<td>1.20 ± 3.1 (range, 0-16.9)</td>
</tr>
<tr>
<td><strong>Cocaine</strong></td>
<td>Positive 28 (26) Negative 58 (53) Indeterminate 23 (21)</td>
</tr>
<tr>
<td><strong>Myoglobinuria</strong></td>
<td>Positive 61 (56) Negative 38 (35) Indeterminate 10 (9)</td>
</tr>
<tr>
<td><strong>Renal failure</strong></td>
<td>Positive 24 (22) Negative 85 (78)</td>
</tr>
<tr>
<td><strong>EKG abnormal</strong></td>
<td>Positive 46 (42) Negative 57 (52) Indeterminate 6 (6)</td>
</tr>
<tr>
<td><strong>Echo abnormal</strong></td>
<td>Positive 20 (18) Negative 43 (39) Indeterminate 46 (42)</td>
</tr>
</tbody>
</table>

Values are expressed as number (%).
study. Data were analyzed using EpiInfo 6 (CDC, Atlanta, Ga) and Microsoft Excel 98.

3. Results

One hundred sixty-three patients were identified by the database. Forty-one patients were excluded for CK less than 1000 U/L and 13 patients were excluded because they did not have a cTnI determination, leaving 109 patients in the final analysis (Fig. 1). The demographics of the study group are summarized in Table 1.

Fifty-five (50%) patients had a positive cTnI. Of the 55 patients with positive cTnI, 32 (58%) were true positives, 18 (33%) were false positives, and 5 (9%) were considered indeterminate. Thus, the prevalence of false-positive cTnI in patients with rhabdomyolysis was 17% (18/109, 95% confidence interval [CI] 0.10-0.25).

Of the 55 patients with positive cTnI, there was no association between false positives and cocaine use, renal failure, or myoglobinuria (RR = 0.95, 95% CI 0.47-1.9; RR = 0.87, 95% CI 0.68-1.7; RR = 1.2, 95% CI 0.64-2.2; respectively). Of all enrolled patients, cocaine use was associated with a reduced risk for a positive cTnI (RR = 0.50, 95% CI 0.27-0.94), whereas renal failure and myoglobinuria were associated with an increased risk for a positive cTnI (RR = 1.45, 95% CI 1.01-2.1, and RR = 2.7, 95% CI 1.5-4.7, respectively). There was poor correlation between peak CK and peak cTnI levels ($r = 0.08$). There were no significant differences between true-positive patients and false-positive patients in peak cTnI (2.2 ± 4.2 vs 1.9 ± 3.4, respectively, $\Delta = 0.29$, 95% CI $-2.1$ to $2.6$) or initial cTnI (0.84 ± 1.5 vs 1.1 ± 3.3, respectively, $\Delta = -0.27$, 95% CI $-1.6$ to 1.1).

4. Discussion

In this retrospective cohort, one half of all ED patients with rhabdomyolysis had positive cTnI, and one third of the patients with positive cTnI were false positives. Thus, one sixth (17%) of all ED patients admitted with rhabdomyolysis had a false-positive cTnI. The range of false-positive cTnI in other studies are similar, ranging from 11% to 35% despite the use of different study methodologies, different assays, and different upper reference limits for cTnI [6,7,11-13].

We examined cocaine use, myoglobinuria, and renal insufficiency in our cohort to determine if false-positive elevations in cTnI were related to these factors. We did not find an association between false-positive cTnI and cocaine use, myoglobinuria, or renal insufficiency, but there were associations between overall positive cTnI and all three factors. Perhaps surprisingly, cocaine use was found to be associated with a decreased risk of a positive cTnI. This finding may be expected as we looked at patients with rhabdomyolysis, not patients with potential acute coronary syndromes, and that patients with cocaine use were significantly younger (39 vs 55 years of age, $\Delta = 16$, 95% CI 8-24). There was a positive association between patients with positive cTnI and impaired renal function. However, the interpretation of elevated cTnI in patients with chronic renal failure remains an unresolved issue, with conflicting results from various studies [15-17]. Finally, one may expect a positive correlation between peak CK and peak cTnI levels in patients with rhabdomyolysis, but we did not find such an association ($r = -0.08$). Our findings were similar to that of Punukollu et al [7], who also did not find a correlation between CK and cTnI ($r^2 = 0.16$).

The meaning of these “false-positive” elevations in cTnI is not completely resolved. The false positives may represent minor cardiac injuries that are undetected by EKG and echocardiography, or they may represent an underlying problem with cTnI assays. Troponin assays are immunoassays, and like all antibody assays, cross-reactions may occur, particularly in patients with rhabdomyolysis in whom the skeletal forms of troponin I are elevated, altering the normal background signal-to-noise ratio [10,18].

There were several limitations in our study. First, our definition of a criterion standard (negative EKG/echocardiography) is disputable. However, such patients with rhabdomyolysis and false-positive cTnI would not satisfy the revised American College of Cardiologists criteria for acute, evolving, or recent MI because they did not have EKG changes and did not undergo revascularization [5]. In addition, no false-positive cTnI patient had an adverse event such as pulmonary edema, arrhythmia, or death while hospitalized. Second, we did not examine patients...
who were discharged from the ED with rhabdomyolysis, and thus, there is selection bias in our patient population. Third, there were several limitations in our study that would lead to an underestimation of the prevalence of false-positive cTnI in patients with rhabdomyolysis. We were extremely stringent in our definition of a false positive. Many of the “abnormal” findings in the EKG and echocardiography were not diagnostic of ischemia, for instance, patients in which chronic atrial fibrillation or global hypokinesis on echocardiography would be considered by many physicians not to be indicative of an acute ischemic event. Thus, many patients who were considered true positives were more likely false positives. In addition, not every patient had an echocardiogram. The prevalence of false-positive cTnI would be higher if any of the 5 indeterminant patients with positive cTnI had a negative echocardiogram. It is likely that these patients did not undergo echocardiography because the treating physicians did not feel that the patients had a cardiac event. Finally, some patients did not have a full “set” of cardiac enzymes; 11% of our cohort had only one or two cTnI determination. Again, it is likely that these patients did not have additional cTnI testing because the treating physicians did not feel that the patients had a cardiac event. We may find more patients with positive cTnI in our cohort if patients had more cTnI testing and may increase the prevalence of false positives.

In summary, ED patients admitted with rhabdomyolysis frequently have positive cTnI, and the prevalence of false-positive cTnI is at least 17%. The false-positive elevations in cTnI are not affected by cocaine use, impaired renal function, or myoglobinuria.

References


Brief Reports

Sustained oxygenation without ventilation in paralyzed pigs with high-flow tracheal oxygen

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Abstract

Objectives: It is generally assumed that ventilation is necessary for oxygenation. This study tested if paralyzed animals without respirations can maintain arterial oxygenation when administered high-flow oxygen delivered by a catheter in the trachea.

Methods: Design: Prospective observational study. Setting: University research laboratory. Participants: 3 anesthetized/paralyzed swine weighing 29.5 ± 4.2 kg. Interventions/observations: Pigs were intubated, anesthetized with intravenous tiletamine and a pentobarbital drip. A femoral arterial line was placed to record arterial blood gases and vital signs every 5 minutes. Respiratory paralysis was obtained with vecuronium 150 µg/kg and repeated at any sign of movement. A catheter was placed in the trachea to deliver oxygen at 15 L/min. Outflow gas from the endotracheal tube was analyzed for O₂ and CO₂. O₂ was discontinued at 75 minutes. The institutional animal care and use committee approved the protocol.

Results: All pigs survived to 75 minutes. PaO₂ was more than 100 mm Hg throughout the study period. Mean PaCO₂ was 37.4 ± 2.8 mm Hg at baseline, 146 ± 59 at 30 minutes, then rose above 200 mm Hg in all pigs by 45 minutes. Mean arterial pH fell from 7.47 ± 0.04 at onset to 6.75 ± 0.06 at 75 minutes. When oxygen was terminated at 75 minutes, PaO₂ fell to 16.5 ± 7.6 mm Hg within 5 minutes, and all pigs were sacrificed within 10 minutes. For outflow gas, O₂ was more than 98% and expired CO₂ less than 1% throughout the study period.

Conclusions: Paralyzed, unventilated pigs receiving high-flow oxygen via a tracheal catheter remained alive after 75 minutes, although a profound respiratory acidosis developed.

1. Introduction

It is generally assumed that ventilation is necessary for oxygenation. In an unrelated study of respiratory paralysis secondary to a toxin, it appeared that the study subjects receiving high-flow oxygen maintained their O₂ saturation as
measured by pulse oximetry after muscle paralysis prevented ventilation. The observation prompted this pilot study to see whether oxygenation can be maintained in anesthetized, paralyzed pigs receiving high-flow oxygen through a catheter inserted into the trachea.

2. Methods

Study design was a prospective observational study. Setting was a university animal research laboratory. Study subjects were 3 domestic swine weighing 29.5 ± 4.2 kg. Subjects were anesthetized with tilamine 5 mg/kg IM. An ear vein catheter was placed, and the pigs were intubated. Anesthesia was maintained with sodium pentobarbital, beginning with an intravenous loading dose of 8.6 mg/kg, followed by a continuous intravenous infusion of 8.6 to 15.2 mg/kg per hour of drip. A catheter was placed in a femoral artery for continuous blood pressure monitoring and to obtain samples for arterial blood gas analysis. Oxygen tubing with internal diameter of 5 mm was threaded through the endotracheal tube so that the end of the tube was at the level of the sternal notch. Oxygen was delivered at a flow rate of 15 L/min. Simultaneous with oxygen delivery, pigs were paralyzed with vecuronium 150 μg/kg. Pigs were continuously monitored for any sign of movement, and the vecuronium dose was repeated whenever any motor activity
was observed. Arterial blood gas samples, pulse, blood pressure, and cardiac rhythm were recorded every 5 minutes. The gas returning from the endotracheal tube was collected in mylar balloons and measured for oxygen and carbon dioxide content at the beginning of the experiment and every 20 minutes. Gas analysis was performed using a True Max 2400 Metabolic Gas Analyzer (Parvo Medics, Salt Lake City, Utah) calibrated with 100% O2. Endpoint was a drop in blood pressure below 15 mm Hg or survival to 75 minutes. Pigs surviving to 75 minutes had the oxygen discontinued, and arterial blood gases were determined every 5 minutes until sacrifice. The protocol was approved by the institutional animal care and use committee.

3. Results

All 3 pigs survived to 75 minutes, although there was no respiratory effort. Oxygenation was maintained (Fig. 1A), although a profound respiratory acidosis developed (Fig. 1B). The arterial blood gas machine only measured PaCO2 levels below 200 mm Hg, and by 45 minutes the PaCO2 was more than 200 mm Hg for all pigs (Fig. 1B). As can be seen in Fig. 1C, the pH had an initial rapid fall and then varied between 6.7 and 6.8 for the final 30 minutes of the experiment.

Hemodynamic changes were dramatic despite sedation with pentobarbitol, with the development of hypertension and tachycardia, as seen in Fig. 2A,B, respectively.

Analysis of expired air indicates that the oxygen concentration remained above 80% throughout the experiment (Fig. 3A). The CO2 content was low, but increased gradually (Fig. 3B), indicating that some respiratory exchange of gas was occurring despite the paralysis of the skeletal muscles. In one pig, a piece of tissue was suspended at the opening of the endotracheal tube. There was a deflection of the tissue coincident with the heart rate, indicating that the motion of the heart was producing air movement into and out of the thoracic cage.

4. Discussion

The most important finding of this study was that high-flow oxygen administered through an endotracheal tube was able to maintain oxygenation of arterial blood and sustain life for 75 minutes despite respiratory muscle paralysis. The progressive increase in expired gas CO2 content, coinciding with the elevation of arterial PaCO2, indicates that some passive gas exchange was occurring despite the paralysis of skeletal muscles.

A profound respiratory acidosis developed. At approximately 30 minutes, the PaCO2 arose above 200 mm Hg, which was the upper limit of detection of our arterial blood gas machine. Arterial pH fell but reached a plateau at approximately 6.8. This may be because a steady state developed, with some carbon dioxide being flushed out of the lungs either through diffusion or taken along with the outflow of oxygen. Hemodynamic changes of tachycardia and hypertension that developed may have been secondary to the profound acidosis.

The mechanism of oxygenation without ventilation is not known. It may be that deoxyhemoglobin in the pulmonary capillary bed has a strong affinity for oxygen and removes oxygen from the alveoli. A diffusion gradient is created, which draws oxygen from the trachea into the alveoli. Alternatively, the movement of the heart and great vessels during cardiac contraction, and the expansion of the pulmonary vessels during ventricular ejection, may compress some portions of the lung. This compression would facilitate air movement over short distances, possibly including the respiratory bronchioles. Consistent with this theory, a small movement of air was noted at the end of the endotracheal tube coinciding with the heart rate in one pig.

The results presented here are consistent with a recent observation by Gupta and Haydock [1], who reported a patient who overdosed on heroin and maintained an oxygen
saturation of 96% and an arterial \( P\text{a}O_2 \) of 176 mm Hg with a respiratory rate of 8, pH of 6.79, and a \( P\text{a}CO_2 \) of 224. This patient was receiving high-flow oxygen by face mask. This case is different from the case reported here in that this patient continued to have some respirations, whereas the pigs had absolutely no respiratory effort.

A computer simulation of hypoventilation [2] found that with high-flow oxygen, \( P\text{a}O_2 \) is maintained in the presence of hypoventilation, although a profound respiratory acidosis develops. These authors found that with a respiratory rate of 0.8 breaths per minute and 100% oxygen delivered by endotracheal tube, \( P\text{a}O_2 \) was predicted to be 127 mm Hg with a \( P\text{a}CO_2 \) of 586 and pH of 6.75. The oxygenation of these pigs was better than this stimulation. After 25 minutes with no ventilation, the \( P\text{a}O_2 \) was 162 ± 28 mm Hg, and pH was 7.01 ± 0.07. Their stimulation was for an individual whose weight was 70 kg and height of 170 cm. The pigs in this experiment were less than half as heavy, weighing 29.5 ± 4.2 kg.

Jet ventilation [3] is a technique to oxygenate individuals in respiratory failure if an endotracheal tube cannot be established. This technique consists of placing a catheter in the trachea through the cricothyroid membrane (trans-tracheal jet ventilation) or through the larynx (translaryngeal jet ventilation). The patient is ventilated with intermittent bursts of oxygen under high pressure that can inflate the lungs. Our procedure was different from jet ventilation in that we did not use high pressure, only 15 L/min from an oxygen tank.

As long ago as 1956, Jacoby et al [4] described 5 patients with airway obstruction from tumors who were scheduled for surgery under general anesthesia. These patients were said to have developed airway obstruction after general anesthesia. A needle of unspecified size was placed in the trachea and used to deliver oxygen at a rate of 4 L/min. Hypoxia was reversed as measured by a pulse oximeter, although arterial blood gas results were not reported. Although \( P\text{a}CO_2 \) was not determined, the authors assumed that a respiratory acidosis occurred because of a mild increase in pulse and blood pressure. These patients differed from the pigs in our study in that they were not paralyzed, continued to have respiratory effort, and the flow of gases from the mouth was noticed during expiration.

The profound tachycardia and hypertension that developed may be secondary to acidosis. Hypertension and tachycardia have been observed as a consequence of experimentally induced acidosis in anesthetized dogs [5]. The changes seen were statistically significant but not as dramatic as those seen here, but the pH was reduced to 7.180 from 7.228 rather than the lower levels that developed in the study reported here.

Apneic ventilation was described, primarily in the Scandinavian anesthesiology literature in the 1980s, as a technique to reduce motion of the thorax during thoracotomy. Studies in humans [6] and dogs [7,8] demonstrated that oxygenation could be maintained with oxygen flow in the absence of chest wall motion. Although we did not know of these experiments before our study and this effect is not widely known in the emergency medicine and resuscitation literature, we believe that these experiments demonstrated the same phenomenon we have observed.

The study presented here has certain limitations. As a pilot study, only 3 subjects were studied. As the data for each pig are so similar to the mean, a small population size is sufficient for our conclusions. The physiological mechanism by which oxygenation occurs without ventilation was not determined. The purpose of this pilot study was to verify a previous observation with monitoring of arterial blood gases.

In conclusion, oxygenation was maintained in these paralyzed subjects with no respirations and high-flow oxygen delivered through a tube inserted in the trachea. A profound respiratory acidosis developed with a concomitant increase in blood pressure and pulse. Further research is needed to determine the utility of this model for resuscitation. This model may have applicability to understanding some aspects of the pathophysiology of resuscitation.

References

Brief Reports

The effect of Combitube use on paramedic experience in endotracheal intubation

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Medical Services, Milwaukee County EMS, Milwaukee, WI 53226, USA

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Abstract
Objective: To determine the change in number of endotracheal intubations per paramedic after the implementation of Combitube use and to explore consequences.
Methods: Before-and-after study; urban/suburban EMS system; population 1 million. Number of patients 16 years and older, type of airway used, attempts, and successes were abstracted for time periods before and after the use of Combitubes. The number of endotracheal intubations/paramedic for each period was calculated.
Results: Three-year pre-Combitube: patients 50,983; 6.6% arrests; 3142 received an endotracheal intubation attempt with 93.5% success. The average annual number of paramedics was 153 with 6.9 ± 6.4 intubations per paramedic per year. Three-year post-Combitube: patients 55,959; 6.0% arrests; 2913 received an advanced airway attempt: 860 Combitubes, success 89.4%; 2144 endotracheal intubations, success 91.6% (95% confidence interval success rate difference, 0.5-3.3; P = .007). The average annual number of paramedics was 177 with 3.7 ± 3.3 intubations per paramedic per year.
Conclusion: After implementation of the Combitube, the number of endotracheal intubation attempts/paramedic and success rates decreased.
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1. Introduction

Airway management has long been a cornerstone of effective pre-hospital care. EMS history is full of debate over the best methods for airway management including which adjuncts are effective and appropriate when used by pre-hospital providers. Although endotracheal intubation is performed by varying levels of pre-hospital providers, this skill is typically limited to providers trained at the advanced life support paramedic level. Given the importance of airway management, many devices have been developed to assist lesser-trained providers in securing an airway. One of these devices, the esophageal tracheal airway (trade name Combitube, Tyco Healthcare Group LP, Mansfield, Mass), is used widely by providers at all levels of training.

Endotracheal intubation is still considered the criterion standard to protect and secure an airway [1]. Intubation is a complex skill in which proficiency is likely linked to
experience. Although the absolute number of intubations per person for maintenance of the skill is unknown and likely differs from person to person, more experience is preferred to less [1,2].

A concern with the introduction of the Combitube in the Milwaukee County EMS system was that the already rare opportunity for endotracheal intubation experience might decrease further. This decrease in experience may reduce paramedic proficiency in endotracheal intubation [3]. The purpose of our study was to determine the change in number of endotracheal intubations per EMT-paramedic per year after the implementation of Combitube use.

2. Methods

This is a before-and-after descriptive study that was awarded exempt status from the Medical College of Wisconsin institutional review board. We used the Milwaukee County EMS Patient Care database that contains information from all advanced life support calls serving this mixed urban/suburban population of approximately 1 million. EMS responds using a tiered model incorporating dual-trained firefighter EMT-basics (EMT-B) and EMT-paramedics (EMT-P). For first-tier responses, EMT-B average response time to a scene is 4 minutes, and EMT-P response time is less than 9 minutes 90% of the time. First-tiered EMT-Bs are trained, equipped, and allowed by standing protocol to place a Combitube in pulseless and apneic patients. Paramedic protocol considers the Combitube as a rescue airway when an endotracheal tube cannot be placed. Rapid Sequence Induction or medication-facilitated intubation is not used in our system.

In Milwaukee County, the first use of a Combitube was documented May 23, 1996. We queried the database for all runs during 1993 through 1995 as our pre-Combitube use control set. We excluded data from 1996, considering it a year of transition, and further queried the database for 1997 through 1999 as our study population, for a total of 6 years. We included patients aged 16 years and older who received an advanced airway attempt with either a standard endotracheal tube or Combitube.

The following data points were abstracted: number of patients seen; type of advanced airway used; number of advanced airway attempts, successes, and failures; and number of intubation attempts performed by each paramedic. Success was defined as a secure and adequate airway as documented by the on-scene paramedic using clinical indicators.

3. Results

During 1993 through 1995, Milwaukee County Paramedics treated 50,983 patients 16 years and older. Cardiac arrest, which was the most common indication for an advanced airway, accounted for 3366 or 6.6% of those patients. Of the cardiac arrests, 358 were traumatic and 3008 medical. During our post-use study period (1997 through 1999), 55,959 patients were seen. A similar percentage of arrests occurred: 3333 patients or 6.0%. The distribution between traumatic and medical causes for the arrest was similar. In the pre-Combitube set, there were 3160 patients on whom an advanced airway was attempted. More than 99% of these advanced airways were with endotracheal tubes. During this period, 18 esophageal obturator airways were used; this airway device was subsequently removed from service before introduction of the Combitube. In the post-Combitube group, 2913 patients received an advanced airway attempt: 2144 endotracheal

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Results</th>
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<tbody>
<tr>
<td></td>
<td>1993-95</td>
</tr>
<tr>
<td>Total patients ≥16 y of age</td>
<td>50,983</td>
</tr>
<tr>
<td>No. of airway attempts</td>
<td>3160 (6.2%)</td>
</tr>
<tr>
<td>ETT attempts</td>
<td>3142 (99.4%)</td>
</tr>
<tr>
<td>ETT success rate*</td>
<td>93.5%</td>
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<td>Combitube attempts</td>
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<td>Combitube success</td>
<td>NA</td>
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<td>Provider placing Combitube</td>
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<td>Total no. of patients w/ advanced airway</td>
<td>2939 (93.0%)</td>
</tr>
<tr>
<td>Total no. of paramedics</td>
<td>459</td>
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<tr>
<td>Avg ETT/EMT-P per year</td>
<td>6.9 ± 6.4</td>
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<td>6.3-7.5</td>
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<td>Max range per year</td>
<td>0-41</td>
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<td>Median per year</td>
<td>5</td>
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<tr>
<td>Avg no. of EMT-Ps with no ETT successes per year</td>
<td>15 (9.8%)</td>
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* P = .007, 95% CI success rate difference (0.5-3.3).
** P < .05.
tubes and 860 Combitubes. The sum of these numbers is greater than the 2913 patients because some patients received attempts with both an endotracheal tube and a Combitube. The endotracheal intubation success rate before Combitube introduction was 93.5%. After the introduction of Combitubes, that success rate dropped to 91.6%. The decrease of 1.9% is statistically significant with a P value of .007 and a 95% confidence interval of 0.5% to 3.3%. The success rate for Combitube placement was 89.4%.

During the pre-Combitube 3-year study period, there was a total of 459 paramedics working, or an average of 153 paramedics per year. Each paramedic had a mean of 6.9 intubations per year with an SD of 6.4 and a range of 0 to 41. For the post-Combitube 3-year study period, there was a total of 531 paramedics, or 177 per year. These paramedics had 3.7 intubations per year with an SD of 3.3 and a range of 0 to 25, a 46% reduction. If we corrected for the increase in the number of paramedics, 153 paramedics from the pre-Combitube study period would have only had an opportunity for an average of 4.7 intubations per year in the post-Combitube period. One hundred thirty-eight paramedics were practicing during both pre- and post-study periods.

In summary, the number of intubations per paramedic decreased, and the range representing the maximum number of intubations a single paramedic performed decreased (see Table 1). The 95% confidence intervals for intubation experience in each period are mutually exclusive. The average number of paramedics without any intubation successes per year increased between the groups from 9.8% to 12.6%.

4. Discussion

In the 1970s, with the birth of modern-day pre-hospital care, debate raged as to whether pre-hospital providers should and could establish a controlled airway via endotracheal intubation. Through that decade, it was shown that paramedics could learn and perform the skill of endotracheal intubation successfully, and intubation has become a standard pre-hospital paramedic skill [4,5]. Still unanswered, however, is the amount of initial and ongoing education, training, and experience that is necessary to maintain proficiency in this skill [2,6]. As controlling the airway has traditionally come first in the ABCs and is paramount in emergency care, airway devices have been developed to help promote more ubiquitous airway control. One such device is the Combitube.

The Combitube’s main advantage is in its purported relative ease of use by lesser-trained providers promoting more rapid airway protection, potentially benefiting a broader range of patient populations in controlling the airway out-of-hospital. The Combitube is also commonly used as a rescue device after a failed standard endotracheal intubation attempt [7].

In this study, it is unclear as to why, despite an increase in patients seen, there is a decrease in the number of patients receiving advanced airway attempts. During this period, there were no protocol changes that would explain the decrease. An astute observer will note that given a decrease in intubation rates, the number of intubations per paramedic must decrease. This mathematical truth, however, does not wholly account for the decrease in intubation attempts. Despite the proven effectiveness of Combitubes [8,9], in our system, any potential benefit of Combitube use may likely be offset by a detrimental decrease in paramedic intubation proficiency. It is essential that new products or procedures that have been shown to be efficacious in the literature not be implemented into an EMS system blindly without examining the patient care consequences. Each intervention should be evaluated to determine its effectiveness in a given system. The use of the Combitube by a first responder as a primary airway may be in question if intubation-proficient second-tier EMT-paramedic’s response is timely.

5. Limitations

We used a retrospective database that always has the risk of being inaccurate or incomplete, but we have no reason to believe a difference exists in the quality of data between the two periods. Second, paramedic attrition and recruitment varied. We did not analyze each individual year with regard to attrition, but more than 75% of the paramedics had patient encounters in both the pre- and post-periods. We caution the reader that we did not establish a cause-and-effect relationship that the introduction of the Combitube was the sole reason for the decrease in intubation success rates. Furthermore, we are still unable to determine the ideal number of intubation experiences required to maintain proficiency.

6. Conclusion

In our EMS system, the number of endotracheal intubation experiences per paramedic was nearly cut in half after introduction of Combitubes into the airway patient care algorithm. The paramedic endotracheal intubation success rate also decreased significantly. Fewer opportunities for endotracheal intubation may further challenge paramedic skill retention and intubation proficiency.

Acknowledgments

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References

Clinical Notes

Correction of factitious hyperkalemia in hemolyzed specimens

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Abstract

Background: Hemolysis in pediatric specimens is common due to difficult blood draws and small-bore intravenous catheters. Values of serum K⁺ become falsely elevated secondary to release of intracellular contents. If a reliable correction factor existed for this factitious elevation, repeat K⁺ measurements might be avoided.

Objective: The aim of the study was to establish a correction factor for factitiously elevated K⁺, using free plasma hemoglobin (p-Hgb) as a measure of in vitro hemolysis.

Methods: Twenty whole-blood specimens drawn from healthy adults via a 23-gauge needle were divided into 4 aliquots: (1) no manipulation, (2) mechanical hemolysis via a 27-gauge needle, (3) addition of potassium acetate (KAc), and (4) addition of KAc and mechanical hemolysis. KAc was added to mimic potentially significant hyperkalemia. All specimens had standard K⁺ and p-Hgb measurements performed.

Results: Nonhemolyzed and hemolyzed K⁺ ranged from 3.2 to 8.1 mEq/L and 3.5 to 10.0 mEq/L, respectively. A linear relationship existed between the change in K⁺ and p-Hgb from the nonhemolyzed to hemolyzed specimens. A correction factor for K⁺ of 0.00319 (95% confidence interval, 0.00290-0.00349) × p-Hgb was obtained.

Conclusions: A reliable correction factor for factitious hyperkalemia in a clinically relevant range exists. By example, using the above correction factor, one can predict that the delta K⁺ in a specimen with 500 mg/dL of p-Hgb will be 1.6 mEq/L (range, 1.5-1.7). We suggest that when the lower bound of the predicted delta K⁺ results in a corrected value within the reference range, a second blood draw is unnecessary.

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1. Introduction

Hemolysis in pediatric specimens is common because of difficult blood draws and small-bore intravenous catheters. Values of serum K⁺ become falsely elevated secondary to
release of intracellular contents which occurs during this mechanical hemolysis. Because plasma hemoglobin is the predominant component that is released, it can serve as the measure of hemolysis. In previous studies, the relationship between hemoglobin and potassium in hemolyzed specimens has been shown to be linear [1].

In one ED study, the time from ordering to reporting of results for serum potassium was 58 minutes with hemolysis of specimens, leading to recollection rates of 1% for venipuncture and 20% for intravenous catheter (nurse technique) because of hemolysis [2]. This results in many repeated blood draws in pediatric patients and prolongs stays in the ED.

A reliable correction factor to correct for spuriously elevated K+ as a result of in vitro hemolysis would allow for physicians to circumvent the costly and time-consuming need to redraw specimens. The current standard is visual grading of hemolysis by laboratory personnel when reporting the results of electrolytes. This visual assessment has been found to be unreliable when compared with spectrophotometric analysis [3,4] and, furthermore, offers no applicable information to the practitioner.

This study was constructed to use free plasma hemoglobin (p-Hgb) as the measure of in vitro hemolysis. This could then be applied to calculate corrected K+ for any given hemolyzed specimen.

2. Methods

2.1. Study design

We performed a controlled trial of the effects of mechanical hemolysis on serum K+ measurements in whole-blood specimens obtained from adult human volunteers. To incorporate the range of serum K+ encountered in clinical practice, the effects of mechanical hemolysis were also studied in specimens to which potassium acetate (KAc) had been added. Approval was obtained from the Colorado Multiple institutional review board, and consent was obtained from each volunteer.

Twenty healthy nonpregnant volunteers aged 18 to 35 years were recruited from among the resident and attending physician staff at a large, tertiary, university-affiliated children’s hospital. All volunteers were believed to be in good health. Specifically, participants were screened for a history of hemolytic anemic conditions or diuretic use. All subjects underwent standard venipuncture in an upper extremity with a 23-gauge needle. Four whole-blood sample aliquots of 3 mL were obtained into 5-mL Vacutainer tubes from each volunteer. The tubes from each participant were categorized into 4 groups as follows:

- **Group 1**: no manipulation
- **Group 2**: mechanical hemolysis
- **Group 3**: addition of KAc
- **Group 4**: addition of KAc and mechanical hemolysis.

Immediately after venipuncture, mechanical hemolysis was induced in groups 2 and 4 by ejecting the aliquot through a 27-gauge needle into a vacuum tube, maximizing surface area by allowing air bubbles to form readily. In each group, the first 10 specimens were passed once through the needle, and the second 10 specimens were passed multiple times. Immediately after venipuncture, KAc (0.054 mol/L) solution was added to the whole-blood specimens in groups 3 and 4. In each of these groups, 25 μL/mL was added to the first 10 specimens, and 50 μL/mL was added to the second 10. P-Hgb was analyzed before and after addition of the KAc to ensure that this process did not evoke hemolysis.

For all specimens, serum K+ was measured on the Dade Behring Dimension RXL by the use of an ion-selective electrode for K+. The p-Hgb was measured on the Varian Dual-Beam Spectrophotometer coupled with a voltmeter for accurate wavelength settings. Calibration and validation of all instruments were in accordance with the institution’s usual standard quality assurance procedures for all clinical specimens.

SPSS 10.0 (SPSS, Chicago, Ill) was implemented to analyze the data. The dependent variable (y-axis) was defined as the difference in K+ measurements (delta K+) between mechanically hemolyzed (groups 2 and 4) and nonmanipulated specimens (groups 1 and 3), resulting in 40 discreet data points. The p-Hgb concentration in the hemolyzed specimens was the independent variable (x-axis). A line of best fit through zero was generated. The slope of the line was the putative “correction factor” for the effect of mechanical hemolysis. A 95% confidence interval (CI) was determined for this correction factor.

3. Results

Significant positive hemolysis interference was found for potassium. As evident from the graph, the increase in plasma K+ in hemolyzed specimens shows a linear relationship to plasma hemoglobin. This relationship holds true in those specimens with minimal or marked hemolysis, although there were not as many severely hemolyzed specimens.

The addition of KAc created specimens which had a higher baseline K+, ranging from 4.8 to 8.1 mEq/L, with most being in a clinically significant range (>5.0 mEq/L). The addition of KAc to those specimens did not evoke hemolysis, with comparable baseline plasma Hgb in those specimen sets without addition of KAc. The amount of mechanically evoked hemolysis was comparable in both the group with and without addition of KAc. There was no significant difference in the amount of hemolysis evoked by mechanical hemolysis in those specimens that were passed once versus those passed multiple times through a 23-gauge needle.
A correction factor for $K^+$ of $0.00319$ (95% CI, $0.00290$-$0.00349$) was obtained from simple linear regression using a best-fit line through zero (Fig. 1).

4. Discussion

A reliable correction factor for factitious hyperkalemia in a clinically relevant range exists. This correction factor is similar to those found in other studies [5]. The method used in previous studies involved preparation of hemolysates on centrifuged and washed whole-blood specimens. The hemolysate was then added back to specimens. This may lead to loss of potassium and perhaps an underestimated correction factor.

A previous study found a correction factor ($0.005$ mEq/mg pHgb) significantly different from that obtained here [6]. The methods involved disrupting a clot with applicator sticks and recentrifuging to obtain clear but hemolyzed serum. A correction factor was obtained by examining the relative error of paired specimens (difference in potassium from nonhemolyzed and hemolyzed specimens divided by serum hemoglobin of the hemolyzed specimen). The average of all correction factors was then computed and reported. This fails to take into consideration a zero point and therefore may overestimate the correction factor. As evident from our plot, our study may have done the same had not a zero point been included.

Our method more closely mimics what occurs in pediatric specimens, that is, the disruption of erythrocytes through small-bore tubing and needles which is a common occurrence. This does not lend itself to error by potassium loss by manipulation of hemolysate. Although the same manipulation did not give consistent results, that was not the goal of his study. We did obtain mechanical hemolysis that gave an appreciable difference in potassium.

By example, using the previously mentioned correction factor, one can predict that the delta $K^+$ in a specimen with 500 mg/dL of pHgb will be $1.6$ mEq/L (95% CI, $1.5$-$1.7$). We suggest that when the lower bound of the 95% CI for the predicted delta $K^+$ results in a corrected value within the reference range, repeat venipuncture is unnecessary. It is important that intravascular hemolysis is excluded either by clinical or laboratory means.

In application, every visibly hemolyzed specimen could be analyzed for hemoglobin concentration. If this was found to be significant, a comment would accompany the $K^+$ results relaying the pHgb results as well as the correction factor to advise clinicians more meaningfully. Alternatively, a program could be set to run the plasma hemoglobin and conduct the correction automatically, and the corrected potassium would be reported to the clinician.

Some clinicians may hesitate to rely on any “correction factor” when the possibility of a life-threatening condition such as hyperkalemia has been raised. We think that such hesitation is unnecessary for 2 reasons. First, in pediatric patients without known renal or endocrinologic disease, true hyperkalemia is rare (ie, pretest probability is low). Second, with the zero point included, the line of best fit for a correction factor on our plot is inherently conservative, with nearly all points falling above the line. Using the lower 95% CI of this factor adds an even greater margin of safety and makes it extremely unlikely that clinically relevant hyperkalemia will be missed.

The cost of the plasma hemoglobin measurements in this system is comparable to a repeat of the potassium
measurement; yet, the variable costs associated with providing a new specimen from the patient are absent. If time spent in the ED is included in these calculations, there are likely cost savings.

Our study aimed to address a clinical issue commonly encountered in pediatric patients; yet, only adult volunteers were used. One can infer that, in the majority of hemolyzed pediatric specimens, intracellular potassium is released by mechanical forces associated with the blood draw. For older infants and adults, the same absorbance properties of hemoglobin exist, which would allow for measurements on the same calibrated spectrophotometer. However, fetal hemoglobin has a different absorption pattern and may be associated with different intracellular release of potassium within whole-blood samples or be associated with different degrees of hemolysis after the addition of KAc. It would be prudent to reproduce the same correction factor taking these factors into consideration in the newborn population.

In this study, there were no true hyperkalemic specimens. We did determine that, in those specimens to which KAc was added, we did not evoke hemolysis by just that intervention. It may prove useful to obtain true hyperkalemic specimens and determine if the same linear relationship holds true. In this study, we were able to evoke hemolysis to obtain borderline but clinically relevant hyperkalemic specimens.

Spectrophotometric analysis of plasma hemoglobin is a relatively inexpensive method of choice. Adjustments would have to be made if samples with fetal hemoglobin are included, as these would have different absorbance characteristics. Our method required 1 mL plasma, which seems reasonable in the pediatric population.

As stated previously, the infant population proves a challenging group to obtain venipuncture specimens. For this reason, conducting the same study and factoring in fetal hemoglobin would be a useful adjunct.

We have shown a linear relationship between p-Hgb and serum potassium which offers a useful correction factor of 0.00319 (95% CI, 0.00290-0.00349) \( \times \) pHgb. This can be applied to many specimens.

References

Wide-complex tachycardia: beyond the traditional differential diagnosis of ventricular tachycardia vs supraventricular tachycardia with aberrant conduction

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Abstract Wide-complex tachycardia (WCT) is defined as a rhythm disturbance with a rate greater than 100 beats/min and a QRS complex duration of 0.12 seconds or more in the adult patient; in the pediatric patient, both rate and QRS complex width are age related. In evaluating this type of tachycardia, there are 2 broad categories usually discussed in the medical literature: ventricular and supraventricular with aberrant intraventricular conduction. There are several other important causes of a WCT encountered in clinical practice, which are less often discussed. These tachycardias require specific therapies differing from the standard approach to WCT. These tachycardias are diverse; as such, the pathophysiology behind each form of WCT includes toxic, metabolic, and conduction system dysfunction mechanisms.

1. Introduction

Wide-complex tachycardia (WCT) is defined as a rhythm disturbance with a rate greater than 100 beats/min and a QRS complex duration of 0.12 seconds or more in the adult patient; in the pediatric patient, both rate and QRS complex width are age related [1,2]. In evaluating this type of tachycardia, there are 2 broad categories usually discussed in the medical literature: ventricular and supraventricular with aberrant intraventricular conduction. A large body of discussion has focused on the 2 major types of WCT: ventricular tachycardia (VT) and paroxysmal supraventricular tachycardia (PSVT) with aberrancy. However, there are several other important causes of a WCT encountered in clinical practice, which are less often discussed. These tachycardias are abnormal, often requiring specific therapies differing from the standard approach to WCT.

In addition to VT and SVT with aberrant conduction, several other causes of WCTs are of importance to the acute care clinician. For example, certain medications produce sodium channel blockade that disrupts intraventricular conduction, resulting in a WCT. Tricyclic antidepressant (TCA) agents may cause serious cardiovascular and neurologic toxicity such as hypotension, arrhythmia, and
seizures. One form of dysrhythmia in this presentation is the WCT [3]. Most patients who develop significant cardiac and neurologic toxicity will demonstrate sinus tachycardia (ST) with QRS complex duration greater than 100 milliseconds, right-axis deviation of 130° to 270°, and/or R wave changes in lead aVR [3].

The antihistamine diphenhydramine (DPH) such as TCA can also be cardiotoxic via its sodium channel blocking capabilities [4]. Blockade of fast sodium channels leads to delayed depolarization and conduction abnormalities. Sodium channel blockade results in a prolongation of phase 0 of the action potential, which will present as prolongation of the PR and QRS intervals. The conduction delay has a greater effect on the right side of the heart as demonstrated by a right-axis deviation in the poisoned patient [4]. Rapid influx of sodium is needed for release of intracellular calcium and myocardial contractility. When bradycardia is accompanied by a wide QRS complex, it suggests severe sodium channel blockade. Besides changes in the electrical conduction, profound sodium channel blockade could result in depressed myocardial contractility, heart block, right-axis deviation, and wide QRS complexes [4].

Another form of WCT is related to the metabolic derangement seen with elevated serum potassium. Hyperkalemia is defined as measured serum potassium of greater than 5.5 mEq/L. Hyperkalemia is characteristically asymptomatic in mild to moderate cases. Its most profound toxicity manifests clinically as altered cardiac conduction that, if unchecked, can progress to life-threatening dysrhythmias [5]. The electrocardiogram

![Wide-complex tachycardia](image)

**Fig. 1** Rhythm strip with wide QRS complex tachycardia in a patient with TCA poisoning.

![12-Lead ECG with a WCT in a TCA-poisoned patient](image)

**Fig. 2** 12-Lead ECG with a WCT in a TCA-poisoned patient. Note the presence of a large S wave in lead I and a prominent R’ wave in lead aVR, findings consistent with TCA cardiotoxicity.
(ECG) will go through a series of changes depending on the serum level. Initially, there will be peaked T waves with prolongation of the PR interval. As the serum level increases, the ECG will progress to loss of P waves, widening of the QRS complex, sine-wave appearance, and, eventually, ventricular fibrillation (VF) and asystole [5]. There is no clear correlation between the degree of hyperkalemia and life-threatening arrhythmias, but arrhythmias are more likely to occur if there is a rapid rise in serum potassium or have serum potassium greater than 6.0 mEq/L [6].

A third form of WCT is seen in certain patients resuscitated from sudden cardiac death. Cardiac arrest continues to be a major public health concern affecting approximately 350,000 people in the United States per year [7,8]. Sudden cardiac death is defined as unexpected natural death from a cardiac cause within a short time, usually 1 hour or less from the onset of symptoms, in a person without any prior condition that would appear fatal [8]. This type of death is attributed to a cardiac arrhythmia, in particular, VT that degenerates to VF [9]. Common postresuscitation dysrhythmias include WCT in addition to other arrhythmias such as narrow complex tachycardia and bradyarrhythmia [10].

Sinus tachycardia, PSVT, and atrial fibrillation (AF) are common dysrhythmias seen in acute care medicine. At times, these tachycardias may present with a wide QRS complex because of a number of pathophysiological events, ranging from preexisting bundle branch block (BBB) and bundle fatigue to ventricular preexcitation, such as Wolff-Parkinson-White (WPW) syndrome. These 3 dysrhythmias all have characteristic electrocardiographic findings and most often present with a narrow QRS complex. With altered intraventricular conduction, the QRS complex will broaden, producing the WCT presentation.

Atrial fibrillation and PSVT with ventricular activation predominantly over the accessory pathway (AP) are common forms of preexcited tachycardia in WPW [2]. Ventricular preexcitation refers to the depolarization of the

![Fig. 3](image)

**Fig. 3** 12-Lead ECG with a WCT. Note the presence of a sine-wave configuration of the QRS complex. This rhythm is an example of the sinoventricular rhythm seen in pronounced hyperkalemia.

![Fig. 4](image)

**Fig. 4** Rhythm strip with a regular wide QRS complex tachycardia his WCT results from a paroxysmal supraventricular tachycardia with aberrant conduction.
ventricular myocardium earlier than would normally occur if the conduction impulses went through the atrioventricular (AV) node. Therefore, there must be another pathway connecting the atria and the ventricles, called the AP. In preexcitation syndromes, the AV node conducts impulses through both the AV node-His pathway and an AP. These 2 pathways provide a reentry circuit that can present as either a narrow or a wide QRS complex. If conduction occurs anterograde via the AV node and retrograde through the AP, the QRS complex is narrow and the pattern is termed orthodromic. If the conduction pattern is opposite (ie, antegrade via the AP and retrograde through the AV node), aberrant conduction occurs, producing a wide QRS complex; this form of conduction is termed antidromic [2].

The goal of this paper is to briefly discuss these not uncommon “other” WCTs.

2. Case presentations

2.1. Case 1

A 26-year-old woman presented to the emergency department (ED) with lethargy and tachycardia. Friends had found her at home with altered mentation. They report that she has been despondent recently over relationship issues. She had no known medical problems and did not use medications. A roommate, however, had “depression” and used a “medication” for this problem. On examination, the patient was lethargic with tachycardia. Other findings included dilated pupils, silent abdomen, and dry mucous membranes. The ECG rhythm strip in lead II (Fig. 1) demonstrated a tachycardia with a wide QRS complex. The 12-lead ECG (Fig. 2) revealed ST with widened QRS complex, prominent R’ wave in lead aVR, and prominent S wave in lead I. Based upon her presentation, the physician considered TCA poisoning. The patient was managed with endotracheal intubation, intravenous sodium bicarbonate, and admission to the intensive care unit. The rhythm diagnosis was a WCT due to sodium channel blockade and tachycardia due to the anticholinergic effects.

2.2. Case 2

A 56-year-old man with known renal failure requiring hemodialysis presented to the ED with weakness and vomiting. He had missed his last dialysis appointment because of severely inelemt weather resulting from a hurricane. He was alert but, when not stimulated, appeared asleep. The 12-lead ECG (Fig. 3) demonstrated a wide QRS complex at a rate of approximately 130 beats/min. With the missed hemodialysis and WCT, hyperkalemia was suspected. The patient was treated with intravenous sodium bicarbonate, calcium gluconate, magnesium sul-
fate, and inhaled albuterol. With treatment, the QRS complex gradually narrowed with a slowing of the ventricular response. The initial rhythm was diagnosed as the sinoventricular rhythm of hyperkalemia.

2.3. Case 3

A 19-year-old man presented to the ED with palpitations. The patient noted associated weakness and fatigue. His medical history was remarkable for corrected congenital heart disease; the specific cardiac lesion and surgical repair, however, were unknown. The examination demonstrated a palpated blood pressure of 95 mm Hg systolic and pulse of 160 beats/min. The ECG rhythm strip (Fig. 4) revealed a WCT at a rate of approximately 160 per minute. The clinician considered both VT and SVT with aberration as a cause of the WCT. Based upon the presentation, the clinician felt that an SVT with aberrancy was the likely rhythm diagnosis. The patient was treated with intravenous adenosine with immediate return to ST; the QRS complex remained widened (Fig. 5). The 12-lead ECG showed an ST with left BBB. Further review of the medical record revealed a history of preexisting BBB, likely because of the past cardiac surgery.

2.4. Case 4

A 19-year-old man called 911 because of near syncope. He noted the sudden onset of palpitations. He had no medical problems and did not use medications or illicit substances. He was very diaphoretic and anxious; no blood pressure was detected and the pulse was rapid at a rate greater than 200 beats/min. Prehospital rhythm strip and 12-lead ECG demonstrated a WCT with rate greater than 250 beats/min and left BBB configuration (Fig. 6). Electrical cardioversion was performed for this unstable WCT. A synchronized cardioversion at 50 J restored ST with adequate blood pressure. He was transported to the ED where a 12-lead ECG demonstrated findings consistent with the WPW syndrome (Fig. 7). The initial dysrhythmia was an AV reciprocating tachycardia seen in WPW patients.

3. Discussion

3.1. Sodium channel blockers

The ability of drugs to block cardiac sodium channels is well known. This sodium channel blockade activity has been described as a membrane-stabilizing effect, a local anesthetic effect, or a quinidine-like effect. 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 depolarization. This creates a delay of sodium entry into the cardiac myocyte during phase 0 of depolarization. As a result, the upslope of depolarization is slowed and the QRS complex widens (Fig. 8).

Myocardial sodium channel blocking drugs comprise a diverse group of pharmaceutical agents (Table 1). As a result, patients poisoned with these agents will have a variety of clinical presentations. For example, cyclic antidepressants, propoxyphene, and cocaine may result in anticholinergic, opioid, and sympathomimetic syndromes, respectively. In addition, these agents may affect not only the myocardial sodium channels, but also other myocardial ion channels such as the calcium influx and potassium efflux channels. This may result in ECG changes and rhythm disturbances not related entirely to the drug’s sodium channel blocking activity.

Sodium channel blockers result in widening of the QRS complex. In some cases, the QRS complexes may take the pattern of recognized BBBs. In severe cases, prolongation of the QRS may result in a sine-wave pattern and eventual asystole. Sodium channel blockers may also induce a monomorphic VT. It has been theorized that the sodium channel blockers can cause slowed intraventricular conduction, unidirectional block, development of a reentrant circuit, and a resulting VT. This can then degenerate into VF. Because many of the sodium channel blocking agents are also anticholinergic or sympathomimetic agents, WCT is commonly seen. However, the sodium channel blocking agents can affect cardiac pacemaker cells. Rarely, bradycardia may occur because of slowed depolarization of pacemaker cells that depend upon entry of sodium. In sodium channel blocker poisoning by anticholinergic and sympathomimetic drugs, the combination of a wide QRS complex and bradycardia is an ominous sign and may indicate that the sodium channel blockade is so profound that a tachycardia cannot be mounted.

A potent sodium channel blocking agent is the TCA responsible for significant human poisoning on a frequent basis [11]. There are 7 major pharmacological effects of TCAs that result in toxicity of the cardiovascular and neurologic systems. Tricyclic antidepressants block the reuptake of biological amines such as norepinephrine, serotonin, and dopamine at presynaptic terminals centrally causing delirium and seizures. Peripheral inhibition produces hypertension and tachycardia [3,12]. In addition, TCA can cause seizures through inhibition of \(^\gamma\)-aminobutyric acid, the primary inhibitory neurotransmitter [13]. Anticholinergic and antihistaminic effects are associated with tachycardia, hyperthermia, and central nervous system stimulation [3]. \(\alpha\)-Adrenergic blockade produces vasodilation and hypotension, which could result in a reflex tachycardia [14]. Potassium efflux blockade prolongs phase 3 of the myocardial action potential, resulting in QT interval prolongation, causing an increased risk for \textit{torsades de pointes} [15]. Tricyclic antidepressants prolong the phase 0 myocardial depolarization by inhibiting the fast sodium channels, resulting in a prolonged QRS complex (Fig. 8) [16].

Antihistamines represent a common toxin seen in clinical medicine with the potential for significant cardiac toxicity, including WCT. According to the 2002 Annual Report of the American Association of Poison Control Centers, DPH was responsible for 28133 toxic exposures, approximately 41% of all antihistamines. Diphenhydramine was also responsible for 12 of 71 reported antihistamine-related deaths, and 18 additional deaths in combination with other medications [11]. Like TCA overdoses, DPH can result in toxicity, including anticholinergic symptoms, central nervous system effects, and cardiac toxicity [4]. The anticholinergic systems often predominate in DPH overdoses. Cardiotoxicity from DPH resembles the effects of TCA agents [17]. Diphenhydramine can result in cardiotoxicity from its ability to block fast sodium channels similarly to Vaughan-Williams type IA antidysrhythmic agents. The ECG changes associated with type IA–induced cardiotoxicity are tachycardia, widened QRS complex, terminal alterations of the QRS complex, and right-axis deviation (R’ wave in lead aVR and S wave in leads I and aVL) [4].

### Table 1  Sodium channel blocking drugs that can cause WCT

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<th>Chloroquine</th>
<th>Class IA antiarrhythmics</th>
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<td>Disopyramide</td>
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<td>Class IC antiarrhythmics</td>
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Wide-complex tachycardia
The sodium channel blocking agents may produce a WCT as seen in Fig. 1. Fig. 9A also demonstrates a WCT seen in a TCA-poisoned patient. Note the prominent S wave in lead I (S1) and R’ wave in lead aVR (RaVR) in Fig. 9B—these findings are strongly suggestive of TCA cardiotoxicity. Note the progressive change of the QRS complex in Fig. 9B with the development of a pronounced R’ wave over approximately 45 minutes in a TCA-poisoned patient.

The management of sodium channel blocking agents consists of administration sodium and/or alkalosis. Infusion of sodium bicarbonate by either intermittent bolus or by continuous infusion has been advocated. Hypertonic sodium infusion has also been shown to be effective. A sodium bicarbonate infusion should be considered if any of the following criteria are met: QRS complex duration greater than 100 ms, persistent hypotension despite adequate hydration occurs, and dysrhythmias. Recall that a single ampoule of sodium bicarbonate contains 50 mEq of sodium, and multiple doses may be necessary to achieve clinical improvement of the patient. Sodium bicarbonate infusions can be created by mixing at least 3 ampules of sodium bicarbonate in 1 L of D5W and infusing twice the patient’s maintenance rate. Potassium should be added (40 mEq/L) to the sodium bicarbonate drip to prevent the development of hypokalemia (due to the excretion of potassium in exchange for hydrogen ions as the kidneys attempt to correct the alkalosis). The infusion can then be adjusted to maintain a blood pH between 7.5 and 7.6. During infusions of sodium bicarbonate, close monitoring of electrolyte, pH, and fluid balance should be performed.

Hyperventilation has been shown to be effective in reversing sodium channel blocking activity, most likely secondary to the induced respiratory alkalosis. Lidocaine has been suggested in the treatment of ventricular dysrhythmias, though clear evidence is lacking. Class IA and IC antiarrhythmics should be avoided because of their ability to block cardiac sodium channels.

3.2. Hyperkalemia

Hyperkalemia is a common electrolyte disorder that can cause lethal cardiac arrhythmia. These arrhythmias are more likely to occur if there is a rapid rise in serum potassium or if the serum potassium level is greater than 6.0 mEq/L [5]. When a patient presents with a WCT, hyperkalemia-related dysrhythmia should be in the differential diagnosis in certain instances (eg, chronic renal failure). In fact, impaired potassium excretion due to renal insufficiency accounts for approximately 80% of the clinically significant hyperkalemia [5]. Another major cause of hyperkalemia is iatrogenic medication (potassium supplementation and potassium-sparing diuretic agents) as well as the excessive use of table salt substitutes [18]. Of course, early in the resuscitative phase of care, these important historical details may not be available to the clinician.

In the resting state, most cardiac cells have membranes only permeable to potassium [19]. Resting membrane depolarization inactivates a portion of sodium channels, reducing the number available to open during an action potential [19]. With progressive hyperkalemia, further resting depolarizations may occur. Eventually, this process results in inactivation of a substantial number of sodium channels; the cell membranes, therefore, are unexcitable, producing bradyarrhythmia and, ultimately, asystole [19]. As atrial myocytes become unexcitable, the P wave amplitude decreases [19]. In the ventricle, sodium channel inactivation depresses intraventricular conduction velocity, producing a widened QRS complex on the ECG (Fig. 3, Fig. 10, and Fig. 11) [19]. The sinoatrial node has reduced sensitivity to hyperkalemia, which means a sinus rhythm can persist and drive the ventricles despite the atrial cells being unexcitable.
As extracellular potassium increases, ventricular myocytes become more depressed and conduction fails at the Purkinje-myocyte junction [19].

In general, potassium levels of 6.5 to 7.5 mEq/L will present with tall peaked T waves (Fig. 11), short QT interval, and prolonged PR interval. Potassium levels between 7.5 and 8.0 mEq/L will have QRS complex widening (Fig. 11) and P wave flattening. Potassium levels between 10 and 12 mEq/L will result in further QRS complex widening and loss of the P wave, producing the sinoventricular rhythm of hyperkalemia (Fig. 3, Fig. 10, and Fig. 11); this progression is soon followed by VF or asystole [20].

The earliest sign of potassium intoxication, hyperkalemia, is the appearance of tall symmetric T waves (Fig. 11); 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. The QT interval may also become shortened at this point. With further increases in the serum concentration, the PR interval is prolonged, followed eventually, and most ominously, with QRS complex widening. Ultimately, in hyperkalemia, the QRS complex assumes a sine-wave configuration and the rhythm is described as sinoventricular (Fig. 3, Fig. 10, and Fig. 11). At any point or time in this pathophysiological worsening, VF may appear. As is true with most physiological derangements, if the process is either slow in progression or long in development, the body is more tolerant of the abnormality.

The resuscitative management of hyperkalemia is guided in a large part by the patient’s clinical situation, including the electrocardiographic findings; in fact, the ECG should guide both the urgency as well as the magnitude of therapy. The goals of the therapy are a reduction of the serum potassium level coupled with a stabilization of the myocardial cell membrane. The serum potassium is temporarily reduced with a transient shift of the electrolyte intracellularly and

![Fig. 10](image_url) Wide-complex tachycardia in a patient with elevated serum potassium. Note the sinusoidal appearance of the QRS complex, consistent with the sinoventricular rhythm.

![Fig. 11](image_url) Severe potassium poisoning in a patient with a serum potassium of 8.7 mEq/d. The initial QRS complex is markedly widened, consistent with the sinoventricular rhythm. With appropriate management, the QRS complex begins to narrow with appearance of a prominent T wave, ultimately forming a normal QRS complex and T wave.
lowered permanently with potassium removal from the body. Response to therapy is often prompt with visualization noted on the electrocardiographic monitor (Fig. 11). The most appropriate initial medication is calcium, delivered in the form of either calcium chloride or calcium gluconate. Calcium works by restoring a more appropriate electrical gradient across the cell membrane; in essence, calcium "fools" the cell into thinking that a more "normal" electrical difference exists between the intracellular and extracellular compartments. Administration of calcium will result in a narrowing of the QRS complex, which is transient, lasting no longer than 30 minutes. Calcium is most appropriately given in the patient with a widened QRS complex. Note that calcium does not cause any shifting of potassium intracellularly. Calcium chloride (13.6 mEq/10 mL) contains significantly larger amounts of calcium per unit volume compared with the gluconate preparation (4.6 mEq/10 mL); calcium should be administered through a large peripheral vein, if possible, in that it is highly sclerosing. The appropriate dose is 10 mL IV over 1 minute in the patient with spontaneous circulation; with cardiac arrest, a similar dose is given via rapid intravenous push. The maximum dose is 20 mL within any given 30-minute period; repeat administrations may be required. Caution is advised in the setting of hyperkalemia related to digoxin toxicity; anecdotal reports suggest an enhanced tendency toward asystole in this clinical setting.

Several agents are capable of transiently moving the potassium from the extracellular to intracellular space; this intracellular shift is short-lived yet will temporarily reduce cardiac irritability and allow a more normalized conduction—essentially, stabilizing the patient—while more definitive therapies are arranged. These medications include glucose, insulin, β-adrenergic agonists, magnesium, sodium bicarbonate, and intravenous saline. Note that the potassium-lowering effect of these various therapies is transient with repeat administration necessary if hemodialysis has not been initiated. Glucose and insulin accomplish the potassium shift via stimulation of the cellular glucose pump. This combination therapy will lower the serum potassium

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**Fig. 12** A WCT in a patient status postdefibrillation for VF. The patient had an adequate blood pressure with this rhythm. This rhythm is actually ST with a widened QRS complex because of bundle dysfunction and ST-segment elevation because of AMI.

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**Fig. 13** The QRS complex gradually narrowed over subsequent minutes with persistent ST-segment elevation consistent with AMI.
level by approximately 1 mEq over 20 to 60 minutes with the greatest reduction occurring in the first 20 minutes. Doses of these 2 agents are as follows: glucose, 50 g IV push, and regular insulin, 10 U IV push. The patient was monitored for hypoglycemia.

β-Adrenergic agonists such as albuterol work via cyclic adenosine monophosphate–mediated potassium pump, resulting in a migration of potassium intracellularly. Albuterol may be given via either intravenous (0.5 mg in 100 mL of saline over 15-30 minutes) or nebulized (10-20 mg in 4 mL of saline over 10 minutes) routes. Albuterol will lower the serum potassium by 1.0 mEq over 30 minutes. Other β-agonist agents such as intravenous epinephrine will have similar effect yet should only be used in a cardiopulmonary arrest scenario.

Intravenous magnesium is another excellent choice, working via stimulation of the sodium potassium adenosine triphosphatase pump. Magnesium produces a rapid reduction in serum potassium, acting as early as 5 minutes after administration. It is given in doses of 1 to 2 g IV over 5 to 20 minutes. The magnitude of the potassium reduction is approximately 0.5 mEq per treatment. Intravenous magnesium also has the added benefit of an antiarrhythmic effect for potassium-related ventricular irritability.

Sodium bicarbonate will promote a shift of potassium intracellularly; it is most appropriately used in patients with acidosis. It should be viewed as an adjunctive therapy to calcium, glucose, insulin, albuterol, and magnesium. Bicarbonate is given 1 mEq/kg IV of body weight; the time of administration ranges from rapid intravenous push in the cardiac arrest and sine-wave QRS complex situations to an infusion over 10 to 20 minutes in the patient with intact circulation. The magnitude of the potassium reduction is best expressed as a function of the pH: for every 0.1 increase in serum pH, the serum potassium should fall by 0.5 mEq.

Intravenous fluids will assist in restoring the appropriate sodium potassium transcellular gradient. It will also induce a diuresis with resultant potassium excretion, assuming that renal function is not excessively compromised. Caution is also advised in patients with compromised left ventricular function.

Fig. 14  A rapid wide QRS complex tachycardia consistent with AF with preexisting BBB.

Fig. 15  Wide-complex tachycardia in a patient with preexisting left BBB. A, Rhythm strip with WCT because of ST with left BBB. B, 12-Lead ECG with left BBB.
Complete and permanent removal of potassium from the body is accomplished via furosemide-hastened saline diuresis, binding resins, and hemodialysis. Binding resins, such as polystyrene, will lower the serum potassium by 0.5 to 1.0 mEq per treatment. The polystyrene may be given either orally, nasogastrically, or rectally in 30 to 60 g treatments. Its effect is slow in onset, requiring 60 to 120 minutes to initiate the process. Hemodialysis is the treatment of choice in such situations and should be used in most of the patients who presented with a sine-wave QRS complex or who have experienced cardiac arrest related to hyperkalemia. Hemodialysis is able to remove up to 50 mEq of potassium per hour of therapy. Peritoneal dialysis may be used yet removes only about 10 mEq of potassium over a much longer period.

3.3. Postresuscitation

Cardiac arrest continues to be a major public health concern affecting approximately 350,000 people in the United States per year [7-9]. Sudden cardiac death is the most common manifestation of coronary heart disease and is responsible for approximately 50% of the mortality from cardiovascular disease in the United States and other developed countries [8,9]. Twenty to forty percent of cardiac arrest is initially resuscitated; however, markedly fewer patients survive to hospital discharge [7]. Common postresuscitation dysrhythmias are WCT, narrow complex tachycardia, and bradycardia [10,21].

The WCT in this instance may result from acute bundle dysfunction related to the cardiac arrest and defibrillatory shocks delivered across the heart. Refer to Figs. 12 and 13 for an example of a patient with acute myocardial infarction (AMI) who was resuscitated from a VF arrest. Immediate postdefibrillation ECG revealed a WCT with adequate perfusion. The QRS complex gradually narrowed over the next several minutes, revealing ST-segment elevation consistent with AMI. The specific mechanism of this WCT is unknown yet may involve acute bundle branch dysfunction with altered inefficient ventricular conduction due to myocardial conduction system “stunning” from the injury. Such a finding usually resolves rapidly after the restoration of spontaneous circulation. This electrocardiographic phenomenon has been observed in patients who have recently undergone electrical cardioversion for dysrhythmia management. Van Gelder et al [22] described this finding in 23 patients who had received electrical

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**Fig. 16**  A, Wide QRS complex tachycardia in an infant with PSVT and rate-related bundle malfunction or bundle fatigue. Note the initial narrow QRS complex that widens as the tachycardia persists (arrows). B, Sinus tachycardia after administration of adenosine in the PSVT patient from Fig. 16. Note the normal QRS complex duration.
cardioversion for AF. They observed this electrocardiographic finding most often in the precordial leads; it was noted less frequently in the inferior and lateral leads, usually concomitantly with anterior elevations. In another report, Madias and Krikelis [23] report 5 patients with AMI complicated by sudden death. In these patients, the typical AMI presentation was described with the early development of either VT or VF. Immediate postdefibril-

**Fig. 17** Irregular wide QRS complex tachycardia in a patient with WPW. Note the beat-to-beat variation in QRS complex configuration and the initial slurring of the QRS complex consistent with the delta wave.

**Fig. 18** Acute myocardial infarction in an adult patient with ST. A, Lead II rhythm strip in a patient with ST-segment elevation. Rapid interpretation of this rhythm strip could lead the clinician in an incorrect path of WCT when, in fact, the rhythm is ST with an elevated ST segment (giant R wave of early AMI). B, Inferolateral AMI.
lotion ECGs demonstrated ST-segment elevation, which persisted for several hours in most instances. The authors noted that these patients suffered “nontransmural myocardial infarction” in most cases, and, therefore, did not attribute this electrocardiographic finding primarily to an acute coronary event.

3.4. Supraventricular tachycardia with aberrant conduction

With aberrantly conducted SVT, the ECG demonstrates the rapid rate and widened QRS complex. The focus of the rhythm is supraventricular, including atrial (ST and AF) and nodal (PSVT) tissues; the electrocardiographic manifestation is tachycardia. The QRS complex is widened because of the aberrant intraventricular conduction; the end result is a wide QRS complex. The aberrant ventricular conduction may be due to a preexisting BBB (Fig. 4 [PSVT], Fig. 14 [AF], and Fig. 15 [ST]), a functional (rate-related) bundle malfunction resulting in a widened QRS complex when the heart rate exceeds a characteristic maximum for that patient (Fig. 16 with PSVT), or accessory AV conduction as encountered in preexcitation syndromes (Figs. 6 [AV reciprocating tachycardia] and 17 [AF]), such as that described by Wolff, Parkinson, and White. Electrocardiographic structures, such as the giant R wave in early ST-segment elevation AMI, may mimic a wide QRS complex; with the development of ST, an apparent WCT will be present (Fig. 18).

3.5. Wide-complex tachycardia with preexcitation syndromes

Wide-complex tachycardia may complicate the WPW presentation in 2 instances, the antidromic form of PSVT, an AV reciprocating tachycardia (Fig. 6), and the aberrantly conducted AF (Fig. 17). The most common rhythm disturbance seen in the WPW syndrome patient is PSVT; this dysrhythmia is termed an AV reciprocating tachycardia. In this instance, activation of the ventricle occurs through the AP. When the impulse loop includes the AP as the anterograde limb and the AV node as the retrograde portion, the resultant QRS complex is wide. With this pathway, the QRS complexes appear wide (essentially, an exaggeration of the delta wave). The ECG demonstrates a very rapid wide-complex tachycardia that is difficult to distinguish from VT. This form of PSVT is referred to as antidromic tachycardia, which is seen in approximately 10% of patients with WPW syndrome. The wide QRS complex results from inefficient conduction of the impulse throughout the ventricle—with an inefficient conduction of the impulse throughout the ventricle, the total time of depolarization is increased, producing a widened QRS complex.

The other form of wide-complex tachycardia seen in this syndrome is AF, representing 20% of symptomatic arrhythmia in WPW. The APs lack the feature of slow decremental conduction; thus, the pathway can conduct atrial beats at a rate that can approach or exceed 300 beats/min, subjecting the ventricle to very rapid rates. The important electrocardiographic clues are the irregularity of the rhythm, the rapid ventricular response (much too rapid for conduction down the AV node), delta wave, and the wide, bizarre QRS complex, signifying conduction down the aberrant pathway. Occasionally, a narrow QRS can be seen, representing conduction through the AV node as seen in the right precordial leads in this example.

4. Conclusion

The emergency physician frequently encounters WCTs. Most often, it is VT, and if the patient is unstable, the patient will need prompt treatment. However, there should be other differential in the context for WCT as well. Sodium channel blocking agents, hyperkalemia, postresuscitation, and AF with BBB all represent a significant minority of WCTs.

References

Controversies

Emergency physicians and the care of children

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Abstract The care of children is an integral aspect of emergency medicine. This article reviews the many important contributions that emergency physicians have made in advancing the acute care of children.

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1. Introduction

Children are frequent users of emergency department (ED) services. Whether for episodic illnesses, acute injuries, or exacerbation of chronic conditions, patients younger than 18 years account for up to 25% of all ED visits in the United States. Despite this large number of pediatric ED visits, controversy persists over the optimal credentials for physicians caring for these patients. Emergency physicians (EPs), pediatricians, and pediatric EPs all routinely treat children in the ED and highlight their training and ongoing patient exposure as justifications for their role in pediatric emergency care. Layered on any professional credentialing controversy are lay publications questioning EPs’ ability to care for children [1-3]. This article discusses the clinical pediatric skills of EPs as well as emergency medicine’s commitment to the practice and advancement of pediatric emergency medicine.

2. Emergency physician pediatric credentials

That emergency physicians (EPs) are key providers of pediatric services cannot be questioned. Recently, the American Academy of Pediatrics (AAP) officially recognized our expertise in treating pediatric patients and endorsed a Society of Academic Emergency Medicine policy stating that EPs possess the knowledge and skills required to provide quality emergency care to children of all ages [4]. The Residency Review Committee of Emergency Medicine (RRC-EM) oversees EM residency program requirements. The RRC is housed within the Accreditation Council for Graduate Medical Education. The RRC-EM’s “Program Requirements for Residency Education in Emergency Medicine” cite the main goal of residency as preparing physicians for the practice of EM [5]. Pediatric EM is considered well within this scope, as emphasized by “The Model of the Clinical Practice of Emergency Medicine” of the American Board of Emergency Medicine [6], in which pediatrics is no longer a separate section of the core content but integral to each. Furthermore, pediatrics is routinely emphasized as a modifying factor for the
American Board of Emergency Medicine oral examination [7]. The RRC-EM demands strict adherence to pediatric training guidelines to ensure appropriate resident exposure to acute pediatric presentations. In preparing residents to provide truly emergent care for patients of all ages, the RRC-EM has established requirements for procedure performance, demanding skill in numerous critical interventions such as airway control and central venous access. Specific guidelines pertain to pediatric emergency stabilization, requiring minimum numbers of pediatric medical and trauma resuscitations.

Residency-trained and/or board-certified EPs now staff most emergency departments (EDs) in the United States, making emergent interventions readily available to pediatric patients in general EDs [8].

Comparisons between EPs, pediatricians, and pediatric EPs have failed to demonstrate any significant or consistent difference in their care of children. Although variations in approaches to patients have been documented in surveys and chart reviews, no outcome difference has ever been identified for any pediatric ED presentation based on the training of treating physicians. More importantly, EPs and pediatric EPs practicing in the same setting develop identical management patterns in their care of children [9-12].

3. Emergency medicine and pediatric preventive care

Advocating for injury and illness prevention remains a top priority in the emergency care of children [13]. Unintentional injuries are the leading cause of death in pediatric patients, and after the first year of life, accidental injuries more than triple the next leading cause of death [14]. Major points of prevention include providing the family with information to help prevent injuries, identifying safety risks and strategies to prevent injury, and serving as a community resource to educate the public, identify hazards, and promote appropriate legislation [15]. The website of the American College of Emergency Physicians (ACEP) contains resources for physicians, including bibliographies for selected pediatric topics, as well as lists of publications and organizations that contribute to injury and illness prevention for children. Emergency physicians are proven leaders in issues of pediatric automobile safety, drowning, bicycle safety, and firearm violence [16-20]. Further injury prevention research will continue to be a priority in pediatric EM. The ACEP Pediatric Emergency Medicine Committee has compiled a summary of the role of EPs in pediatric injury prevention, which serves as a thorough reference [21].

4. A decade of advancements in pediatric emergency care

Emergency physicians have a duty to advance the care of children [22]. Through active participation, EPs have promoted policies and guidelines for pediatric emergency care in the prehospital and hospital arenas, collaborating on projects with the AAP, the American College of Surgeons, the National Emergency Medical Services for Children (EMSC) program of the Maternal and Child Health Bureau, and the National Highway Traffic Safety Administration.
Specific contributions to the field of pediatric EM are highlighted below.

5. Review of policies and guidelines dealing with hospital preparedness for children

In 1993, the ACEP Pediatric Emergency Medicine Committee developed a policy resource and education paper entitled “Report of Preparedness of the Emergency Department for the Care of Children.” In 2001, key aspects from this pivotal policy were then incorporated into a joint policy statement with the AAP [23-25]. These articles have been of value to EDs throughout the country in updating their pediatric equipment so that they are adequately equipped and prepared to handle critical pediatric patients.

It is the EPs’ responsibility to educate and provide medical direction to out-of-hospital providers in addition to participating in emergency medical service quality improvement programs and legislative activities promoting EMSC and injury prevention initiatives. Several important policies and guidelines have been implemented to facilitate and improve the care of children before their arrival in our EDs. The ACEP and the American College of Surgeons have established a joint policy on pediatric equipment for ambulances. Several articles have been published by EPs on this topic [26-28]. In 2001, the ACEP published “The Role of Emergency Physicians in Emergency Medical Services for Children” [29]. A supporting article on the importance of prehospital care in the care of children was published in September 2003 [30]. These articles recognize the important role of EPs in providing medical direction to local, regional, state, and national emergency medical service and EMSC systems.

6. Policies and guidelines regarding the care of children

Emergency physicians have contributed greatly to the current standards of care regarding children with acute diseases. The Broselow-Luten length-based resuscitation tape was created by 2 EPs and has revolutionized the care of critically ill children. Table 1 shows many of the important articles and policies that have been published or developed regarding pediatric clinical care. The policy statements have been adopted by the ACEP Board of Directors and are official policy statements of the college. They are available for review on the ACEP website (www.acep.org).

7. Continuing medical education activities focused on pediatric emergency care

The comprehensive course APLS (Advanced Pediatric Life Support): The Pediatric Emergency Medicine Resource was developed in a collaborative effort between the ACEP and the AAP. This course has recently been redesigned and can be used for training purposes in both pediatric and EM training programs. Both the American Academy of Emergency Physicians and the ACEP are also dedicated to providing pediatric-specific educational opportunities and offering lectures on pediatric EM at both their national and international EM conferences.

8. Conclusion

Emergency medicine practitioners are trained to evaluate and treat all types of emergencies, including pediatric emergencies. The public should feel comfortable in the knowledge that EP training has provided for the emergency evaluation and treatment of pediatric patients as well as patients with medical or surgical illnesses/injuries [45].

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Globalization, digitization, and emergency medicine

As the American Journal of Emergency Medicine (AJEM) approaches its 25th anniversary of publication, the world of publishing has evolved greatly, arguably much more so than the specialty of emergency medicine itself. Medical publication and production are consolidating as an inevitable response to globalization, the digital era of communication, and the information explosion. In the last 5 years alone, AJEM's international submissions and foreign readership have skyrocketed. Researchers in even the most remote countries depend upon worldwide distribution of every journal; English has truly become the lingua franca of science and medicine (witnessed by the large growth in Chinese published journals in English in the past decade), and clinical medicine is becoming an increasingly interdisciplinary exercise with growing overlap between fields as well as between basic and applied research. In 2004, there were more than 20000 science and technology journals being published, with nearly a million and a half articles published each year.

The centuries old, traditional publishing cycle and processes are simply inadequate to address these changes. Yet, medical journals must continue to make major contribution to the progress and application of science, enhance health care through the role of the literature, disseminate research results effectively, and provide relevant information solutions. In addition, they must enhance the peer review process to wade through the bewildering number of submissions; AJEM alone received more than 100 papers last month to evaluate, far more than it can possibly publish in “hard copy.” Preservation of academic integrity and the growing archival and search needs are also pressing issues. Most significantly, because the evidence-based medicine movement begins to acquire momentum, the literature must be widely available and readily searchable to contribute to enhanced quality of care and the elimination of excess costs in health-care delivery.

In response to these changes, AJEM has developed a completely Web-based platform for manuscript submission, review, revision, and editing (the gateway is located at www.ees.elsevier.com/ajem). This will allow us to seamlessly link reviewers and authors in an increasingly rapid, focused, and specific fashion. Reviews will commence within days, even hours, and response time will be limited more by Internet access than any other factor. Beta testing over the first part of the year indicated that more than 95% of our authors and reviewers now have broadband access, rejection rates (already exceeding 66%) will increase, and that overall editorial and revision turnaround times (already rapid) will be cut well more than 50%. Accordingly, AJEM’s digital submission and review process went live on June 1, 2005. Reviewers and readers will both benefit greatly from these enhancements, and all manuscripts will now be submitted, reviewed, edited, and published digitally.

Because the inevitable rollout of health information technology transforms the delivery of health care, it will be important for medical journals to remain technologically relevant to maintain their ability to enhance health care. Emergency medicine has always been at the cutting edge of information technology, and it is most appropriate that its literature migrate into this new era as well.

J. Douglas White, MD, MPH
Editor
We have a shortage of specialists

In economics, a free market forms an equilibrium price of an entity based on supply and demand. If the supply is below this equilibrium point, then the price of the entity increases to stabilize the market. However, if artificial forces intervene, then what develops is a shortage (if the government regulates the price by creating a price ceiling below the market price) or a surplus (if the government creates a price floor or provides subsidies to keep the price higher than the market price).

For medicine in general, specifically to emergency medical care, we are experiencing a shortage of many specialists [1-7]. For trauma care, the difficulty in maintaining call panels for surgical specialists is critical. Prices (reimbursement to physicians and hospitals) are regulated by insurance companies and the federal government, effectively setting a price ceiling. Limited supply, great demand, and a price ceiling fulfill all the economic prerequisites for a shortage. The consequences of this shortage are most critical with the surgical specialists where there is a limited ability for substitution to apply (eg, 1000 internists cannot substitute for a neurosurgeon if the patient requires surgery in the brain, and 1000 pediatricians cannot substitute for an orthopedic surgeon if the patient has an open fracture).

Many of us have had difficulties in obtaining the services of a surgeon or specialists. Emergency physicians can be considered large consumers or “commodity brokers” of the services provided by surgeons and specialists. We have customers (patients) who need specialists and it is part of our job to find one. Finding one during a shortage is inevitably difficult.

The supply of specialists is dependent on training programs, most of which have a focused concern on the resident and fellow workforce needs of the training center itself. Resident and fellow training programs are expensive even with government subsidies for graduate medical education. Insurance companies do not usually cover the cost of graduate medical education directly. In addition, specialty organizations will often stipulate workforce requirements per capita to optimize the distribution of specialists in regions across the country. Although it would be a waste of resources if too many specialists were trained and underemployed, potentially increasing the likelihood of unnecessary surgical procedures, a balance of specialists is required, but above all, when a patient needs the expertise of a specialist, one must be available.

Anticipating the number of specialists per capita is difficult. The tendency of a specialty society is to err on the low side because a surplus must be avoided. This would unfavorably compromise the economics of the specialists that are represented by the specialty organization. Factors adding to the difficulty of predicting specialists per capita include the variability in productivity of each physician. This is true for all physicians in that some physicians are willing to work at a 1.5 full-time equivalent (FTE), whereas others are willing to work at a 0.5 FTE. Family, childbirth, child rearing, lifestyle factors, malpractice risk, commuting distances, academic and research endeavors, and the health status of physicians are some of the factors that affect the FTE productivity of a physician. Geographic proximity permits the sharing of specialists, but this is not as useful for remote areas such as Alaska and Hawaii. Some sparsely populated portions of the lower 48 states have similar geographic difficulties as well. Even if the country as a whole has enough specialists, geographic maldistribution of specialists results in shortages in some areas. For subspecialists, vacations or the attendance of specialty society and educational meetings result in temporary shortages when 1 or more of the specialists must leave town.

The first step to addressing the shortage of physician specialists is to acknowledge that the shortage exists. We must acknowledge that the problem will not be solved without active intervention.

Free market pricing in health-care economics, in which a price equilibrium is determined by supply and demand, is not possible. The economics of health care are highly regulated; there is no free market. Because patients pay only a fraction or none of the charges in health care, the normal economic effect of the price on demand does not exist. If the consumers do not know or do not care about the actual cost of medical care, there is no price effect. Furthermore, prices are regulated by insurance companies and the government.
further eliminating any effect that price might have on the economic equilibrium.

Many hospitals have attempted to modify the price factor by paying specialists to take call. It is common for some physicians to be paid $1000/d just to be available. But in many instances, such subsidies fail to maintain consistent call coverage. Surely, if subsidies fail to address the shortage, this indicates that the shortage is severe.

The ability to substitute 1 surgical specialist for another can help to cover a shortage; however, this substitutability factor is becoming more limited. Specialists perform procedures that are more sophisticated than ever before. The ability for generalists or related specialists to perform these procedures is therefore more limited. Malpractice liability has also created a legal standard in which any procedure must be performed at the standard of care, which is generally set at the level of the specialist. The training of medical and surgical specialists have new limits on the actual hours spent in training, which limits their experience and training time. Specialists of a previous generation could perform many subspecialty procedures. This is less likely today.

A physician is trained to render care. If there is a shortage of specialists in a community, there is an obligation felt by the existing specialists to provide these services, but at some point, they have other obligations (eg, sleep, their practice, their family, and others). Volunteerism will not eliminate the shortage in the long run.

Specialty training programs must recognize the shortage and train more specialists. Insurance companies and the federal government must provide the funds required to train these specialists. Patients are the subscribers to these insurance plans and the recipients of federal health-care funding. If patients need specialists who are in short supply, then the insurance companies and the federal government should provide the funding assistance needed to eliminate the shortage.

However, economics creates a conflict of interest because specialty training programs are run by the specialists who economically benefit from the short supply. From a provider’s (specialist’s) standpoint, a shortage is economically preferable to a surplus. Patients tend not to advocate for themselves, thus insurance companies and the government must represent them and advocate for more specialists to balance the economic forces. However, a shortage results in rationing, which reduces the cost of care for insurance companies and the federal government.

The status quo is maintained because the shortage is economically advantageous to existing specialists, insurance companies, and the federal government. Patients are the victims, but they have not exercised their power to modify the status quo. The status quo can be modified by increasing the supply of specialists.

Medical schools that oversee training programs can take an active role in increasing the number of specialty training slots in their training programs. One of the missions of a medical school is to provide for the medical personnel needs of a community. Small medical schools serve specific smaller communities. But larger medical schools, especially state-supported ones, have larger communities to serve. Medical schools should recognize the shortage of specialists and address this by increasing the size and scope of their specialty training programs. When there are national shortages, this ultimately serves patients by permitting them to obtain optimal quality care in a timely fashion.

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References
Case Reports

Computed tomography scan for the diagnosis of esophageal foreign body

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The individual with the complaint of a bone stuck in the throat is not an uncommon visitor to the ED. An evaluation of such a patient generally includes plain films of the neck and flexible laryngoscopy. If these tests are unrevealing, the patient is often sent home with the assumption that the foreign body has passed with any residual discomfort being attributed to an abrasion resulting from the passage of the bone. We report a case of a man in whom an ingested chicken bone was not identified on multiple soft tissue x-rays as well as flexible laryngoscopy, but ultimately identified with a computed tomography (CT) scan on a return visit to the ED.

An 87-year-old edentulous man presented to the ED with dysphagia, stating that he had accidentally swallowed a chicken bone 3 days earlier. Plain radiographs, including standard frontal and lateral views of the chest and anteroposterior and lateral soft tissue views of the neck, did not reveal a foreign body or soft tissue swelling. A flexible laryngoscopy was performed which showed no evidence of a foreign body or other abnormality. The patient was discharged under the assumption that the chicken bone had passed spontaneously.

Ten days later, he returned to the ED complaining of shaking chills and odynophagia that had progressed from dysphagia. He was reevaluated with anteroposterior and lateral soft tissue views of the neck, which again did not identify any foreign body or soft tissue swelling. Before a planned upper endoscopy, a CT scan of the neck and thorax was performed without intravenous contrast at 3-mm intervals with a HiSpeed CT scanner (General Electric Medical Systems, Milwaukee, WI). The study revealed a proximal esophageal radiopaque foreign body with a transverse orientation, just inferior to the level of the cricoid cartilage, measuring 2.8 × 1.1 cm with intraluminal and extraluminal portions. Edema of the esophageal wall was also present in the area adjacent to the foreign body without evidence of perforation (Fig. 1). Having definitively identified the presence and location of a foreign body, the patient was taken to the operating room, where a V-shaped 2.5 × 0.5 × 0.2 cm chicken bone was removed from the esophagus under general anesthesia with rigid esophagoscopy (Fig. 2). His postoperative course was unremarkable.

Fig. 1 Axial noncontrast CT scan shows proximal esophageal foreign body (white arrow).
Evaluation of esophageal foreign bodies has been conducted using a wide variety of techniques, both radiological and endoscopic [1]. Plain radiography, with standard frontal and lateral views of the chest and lateral soft tissue views of the neck, is the screening technique of choice because of its ability to reveal most true foreign objects and free air in conjunction with low cost and easy accessibility. However, fish or chicken bones, wood, and thin metal objects are often not readily seen with plain radiography. Indeed, plain radiography was unable to identify a relatively large chicken bone in our patient on 2 separate occasions.

Some authorities recommend the use of a contrast esophagram to clarify the presence or location of a foreign body if symptoms are not clear or specific. However, this technique is problematic because it may interfere with future endoscopy. Because of the increased risk of perforation in the setting of untreated esophageal foreign body, it is recommended that persistent symptoms related to the esophagus or trachea should be pursued with direct visualization (ie, endoscopy and/or laryngoscopy) even after an apparently negative radiographic examination [1].

Although laryngoscopy and flexible endoscopy can be helpful in the identification of foreign bodies, their utility may be limited by several drawbacks. Although the airway and upper gastrointestinal tract can be well visualized, it is not possible to assess the surrounding soft tissues to rule out a migrated foreign body, as was the case in our patient. A recent study showed helical CT both sensitive and specific in the evaluation of foreign bodies in the upper esophagus [2]. Three-dimensional CT has also been used successfully to better identify the location of lower gastrointestinal tract foreign bodies in the preoperative setting [3].

CT has many qualities that are advantageous in the primary diagnosis of foreign bodies. A noncontrast CT scan can be performed rapidly in most EDs without the need for sedation or the availability of an experienced operator, as opposed to laryngoscopy and endoscopy. CT can also provide valuable information not only about the presence of a foreign body, but also about its precise location and the condition of surrounding structures and soft tissues. Our experience and the literature suggest that, in problematic cases of foreign body ingestion, CT should be considered as the screening technique of choice. By combining ready accessibility with the ability to provide detailed anatomical information, CT can help to rapidly render a diagnosis and to guide effective treatment.

References

Emergent hemodialysis for acyclovir toxicity

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Acyclovir (ACV) is an effective antiviral agent for remedying herpes simplex virus and varicella-zoster virus infection. Nephrotoxicity, caused by crystallization of ACV and intratubular obstruction, was a well-known toxicity of ACV. In contrast, neurotoxicity, presented as confusion, hallucinations, seizures, and coma, is rarely encountered. Clinically, these neurological symptoms have been mis-interpreted as symptoms of herpes encephalitis [1]. Under this circumstance, physicians tend to increase ACV dosage in fear of disease progression. We report a case who received treatment of ACV then developed concurrent acute renal failure and neurotoxicity. Her neurological symptoms dramatically improved after emergent hemodialysis received in the emergency department (ED). We emphasize the dual role of emergent hemodialysis as a diagnostic tool in the differential diagnosis between ACV-induced neurotoxicity and herpes encephalitis, as well as a fast and reliable treatment of drug-induced neurotoxicity.

A previously healthy 59-year-old woman had right eye pain for 3 days after vesicle formation at the forehead. Under the impression of right herpes zoster involving the first branch of trigeminal nerve and eye, she received intravenous ACV, 250 mg every 8 hours, at a local hospital. Her initial laboratory data revealed normal complete blood counts. Her blood urea nitrogen (BUN) was 6 mg/dL, serum creatinine (SCr) was 0.6 mg/dL, and sodium level was 135 mEq/L. Unfortunately, 2 days later, she became drowsy and lethargic, with incoherent speech and hallucinations. She was immediately transferred to our ED. Upon arrival, her blood pressure was 181/117 mm Hg, body temperature was 37.1°C, heart rate was 128 beats per minute, and respiratory rate was 18 breaths per minute. Although oriented to places and persons, she was still drowsy and agitated. Her pupil size was 3 mm symmetrically with prompt light reflex. Neurological examinations revealed that muscle strength of the extremities was more than grade 4 symmetrically, and the deep tendon reflex was grade 2 to 3 with negative Babinski reflex bilaterally. Laboratory studies found that BUN was 45.1 mg/dL (4.5-24 mg/dL), SCr was 5.0 mg/dL (0.6-1.3 mg/dL), and sodium level was 121 mmol/L (135-148 mmol/L). Magnetic resonance imaging (MRI) of the head was normal. Lumbar puncture was refused by the family. Electroencephalography (EEG) showed mild diffuse cortical dysfunction with more emphasis in the right hemisphere and regional epileptiform activities in the bilateral frontal and the right parietal region. Because of deterioration of renal function and debilitating nature of neurological symptoms suspected due to ACV intoxication, hemodialysis was initiated. The pre-hemodialysis plasma ACV concentration at trough level was 18 mg/L. The therapeutic peak and trough concentrations ranged from 5.5 to 13.8 mg/L and 0.2 to 1 mg/L, respectively, in adults who received 5 mg/kg of ACV [2]. The patient underwent two sessions of 4-hour hemodialysis in 2 days. The post-hemodialysis plasma ACV level was
decreased to 3 mg/L. Her consciousness became clear and BUN and SCr were 17.1 and 1.6 mg/dL, respectively. She was discharged on the fifth day after visiting ED.

There had been various reports on the common side effects such as nephrotoxicity and neurotoxicity in patients receiving ACV [3]. Clinically, ACV-induced neurotoxicity is often similar with herpes-associated encephalitis. Often, emergency physicians may face challenges in differentiating these diseases and the dilemma of whether to continue the medication. Clinically, both herpes-associated encephalitis and ACV-induced neurotoxicity can be presented as subtle change of conscious level, ranging from delirium to coma [4]. Sudden onset in symptoms, absence of fever and headache, and lack of focal neurological finding are more frequently encountered in ACV toxicity [4]. Unfortunately, these clinical findings are not specific. Lumbar puncture, neurological images, and EEG sometimes can aid us during differential diagnosis. Although lumbar puncture is an important test to diagnose herpes encephalitis, about 5% of patients with viral encephalitis have a normal CSF profile, which is likely to be confused with ACV neurotoxicity [5]. Brain computed tomography (CT) may be normal in viral encephalitis, especially early in the illness. Brain MRI is sensitive in the early stages of viral encephalitis, although, rarely, it may be normal in this condition [6]. In cases of ACV neurotoxicity, both CT and MRI are normal. Electroencephalography is particularly useful in the diagnosis of encephalitis showing various forms of discharges that are not specific to the disease and can be seen in cases of ACV neurotoxicity [5]. Therefore, these examinations are useful but not specific to differentiate between the two diseases.

Discontinuation of ACV and removal of the drug by hemodialysis have proven to be the most effective treatment for neurotoxicity [7]. Furthermore, emergent hemodialysis can be a rapid way to differentiate between symptoms of encephalitis and ACV toxicity [8]. By lowering the plasma ACV level, hemodialysis can restore the renal function and consciousness level within hours.

In conclusion, the dilemma of rapidly diagnosing cases of ACV neurotoxicity lies on the need for a specific diagnostic tool that is readily available in all EDs. Emergent hemodialysis is a valuable tool for the early diagnosis and treatment of ACV neurotoxicity.

References

Intracranial foreign bodies related to trauma have been well described over the centuries. King Henry II of France died in 1559 after a broken shaft of a lance was driven through his orbit during a joust [1]. The severity of the potential complications arising from intracranial-penetrating foreign bodies is often quite dramatic and includes intracranial hemorrhage, major vessel injury, arteriovenous fistula formation, focal neurological deficits, cerebral contusion and edema, hydrocephalus, seizures, and central nervous system infections with the majority of deaths generally occurring early from intracranial bleeding [2]. Many penetrating intracranial foreign bodies are simple to identify from external trauma. Occasionally, diagnosis may be difficult with more occult objects or routes of penetration [3-9]. We present a case which presented to our institution with delay in diagnosis.

A 27-year-old white man presented to an outside emergency room after being hit with a branch in the face while riding a horse. The patient was noted to have an approximately 2-cm laceration of the upper eyelid which was primarily closed without complications. On the next day, the patient presented to the emergency room again complaining of headaches, fever, and decreased vision in the affected eye. A computed tomography scan of the brain was performed which revealed a foreign body tracking through the ethmoid and sphenoid sinuses and extending intracranially (Fig. 1). He was diagnosed with meningitis and retained intracranial foreign body. An ophthalmologic examination revealed decreased range of motion of the left eye and 20/70 vision in that eye, which was worse than baseline. An angiogram was obtained which showed no carotid artery abnormalities. He was started on intravenous antibiotics and taken to the operating room for removal of the foreign body. The patient’s traumatic laceration was opened, and a wooden foreign body was noted approximately 1 cm under the skin. The wound was explored, and a
7-cm wooden stick was removed. The patient was taken to the intensive care unit, and his postoperative course was complicated by syndrome of inappropriate secretion of antidiuretic hormone which resolved after medical treatment. The patient was discharged with resolution of his syndrome of inappropriate secretion of antidiuretic hormone and improvement of his vision. In follow-up, the patient’s vision had returned to preoperative levels, and he had no long-term neurological sequelae.

1. Discussion

A majority of penetrating intracranial trauma is due to high-velocity injuries, often gunshots, with obvious diagnosis from external signs [5]. In some instances though, intracranial foreign bodies are less obvious and can result in a delayed diagnosis. In addition, plain radiographs of the head will not reveal radiolucent objects such as wood and plastic. Delay in diagnosis of intracranial foreign body has been reported as long as 15 years after the initial incident [4]. A study by Miller et al found that, in patients with intracranial wooden foreign bodies, survival was only 38% without surgery but improved to 90% with a combination of appropriate antibiotics and surgery [10]. A high index of suspicion is warranted in any patients presenting with a history of what may appear to be minor facial trauma but with a symptom complex out of proportion to physical findings.

References

Case Reports

Diagnostic pitfall: carbon monoxide poisoning mimicking hyperventilation syndrome

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Carbon monoxide is a great imitator. It is colorless, odorless, and ubiquitous in our environment. In large concentrations, it is known to be a stealthy killer. In lower concentrations, patients with carbon monoxide toxicity often are unaware of having had carbon monoxide exposure and may simply complain of vague and varied flu-like symptoms (headache, dizziness, nausea, vomiting, diarrhea, weakness, or general malaise) [1]. We report a case of a young female with carbon monoxide poisoning, who initially presented with delirium, breathlessness, carpopedal spasms, and numbness of the hands and around the mouth. She was therefore initially misdiagnosed as having hyperventilation syndrome. This unusual presentation of carbon monoxide intoxication delayed correct diagnosis and therefore specific treatment in this patient. We were able to find only 1 previous report of a similar case of carbon monoxide poisoning presenting as hyperventilation syndrome in literature [2].

A 23-year-old female with symptoms of shortness of breath and confusion was brought by ambulance to our ED. Her roommate had contacted police after entering the house when she noticed that the patient was delirious and sitting naked on the floor outside the bathroom. The police officers found that the patient was frightened, tachypneic, and unable to talk when they arrived. On their way to the hospital, the roommate was told to hold a paper bag tightly around the patient’s mouth for several minutes.

On arrival in the ED (about 30 minutes later), the patient was still hyperventilating but was able to talk. She denied taking illicit drugs or alcohol but recalled seeing a ghost while taking bath. Her vital signs were blood pressure of 105/70 mm Hg; pulse rate, 126 beats per minute (regular); respiratory rate, 36 breaths per minute; and temperature, 36.2°C (97.2°F). Auscultation of the chest gave normal results. She had numbness in both hands and around the mouth. She also had cramps over her hands and feet. However, no focal neurologic signs were elicited. Arterial blood gases revealed pH 7.51; PaCO₂, 22 mm Hg; and PaO₂, 96 mm Hg (room air). The presumptive diagnosis was panic disorder with hyperventilation syndrome. She was instructed to breathe intermittently into a paper bag. The toxicological screen was negative, and a psychiatrist was consulted to evaluate her visual hallucinations. During her interview with the psychiatrist, the patient volunteered that she had a new gas water heater installed indoor and she had closed all the external doors and windows of the house while bathing. Without delay, she was given 100% oxygen using a tight-fitting face mask with a reservoir bag because carbon monoxide poisoning was suspected. The diagnosis was then confirmed by measuring her blood carboxyhemoglobin level, which turned out to be 28.8% (about 100 minutes

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after removal from scene). She made a full recovery after 22 hours of normobaric oxygen therapy in the ED. No delayed neurologic sequela was found during her scheduled visit to the neurology clinic 4 weeks after the event.

Exposure to fires in closed spaces, vehicle exhaust fumes, combustion fumes, and vapors of paint removers containing methylene chloride can all lead to carbon monoxide poisoning. The clinical presentation of carbon monoxide poisoning is extremely variable. Common symptoms of exposure to low concentrations of carbon monoxide include headache, fatigue, confusion, dizziness, paresthesias, chest pain, palpitations, visual disturbances, and gastrointestinal symptoms [3].

The diagnosis of carbon monoxide exposure requires careful history taking, astute physical examination, and in particular, a high index of suspicion [4]. The diagnosis is confirmed by measurement of blood carboxyhemoglobin. Carbon monoxide alters the dissociation properties of hemoglobin and reduces oxygen delivery to tissues, leading to central hyperventilation and respiratory alkalosis. As a result, characteristic symptoms of hyperventilation syndrome such as shortness of breath, lightheadedness, fatigue, numbness, carpopedal spasms, and fainting spells can also be found in patients with carbon monoxide poisoning.

The cornerstone of treatment of carbon monoxide poisoning is supplemental oxygen, which hastens the dissociation of carbon monoxide from hemoproteins in direct relation to the partial pressure of oxygen. The outcomes of previous studies comparing hyperbaric oxygen (HBO) and normobaric oxygen therapy have been inconclusive because of methodologic difficulties. Although most reported nonrandomized studies have suggested benefit from HBO, results of published randomized studies remain controversial [5-8] Finally, it must be emphasized that neither HBO nor any other therapy can be expected to prevent cognitive deficits due to cell death sustained during the episode of poisoning. [9] Therefore, prevention of carbon monoxide exposure remains the most important means to reduce the morbidity and mortality associated with carbon monoxide exposure.

In conclusion, this case has brought to our attention that carbon monoxide poisoning can be misidentified by health personnel as psychogenic hyperventilatory attacks. We hope that a heightened awareness of this situation by emergency physicians, especially in winter months, will improve the diagnosis and management of this life-threatening condition.

References

Case Reports

Posttraumatic cervical disc herniation: an unusual cause of near drowning

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1. Introduction

Drowning and near drowning are important causes of morbidity and mortality in young people \cite{1,2}. Drowning risk varies widely by geography and patient age. Most submersion events occur in freshwater such as swimming pools, lakes, rivers, ponds, ditches, bathtubs, and so on \cite{3}; however, drowning can occur in seawater.

Cervical injuries are the most common associated injuries in patients with drowning; these spine injuries occur in approximately 0.5\% to 5\% of drowning cases, and they are most commonly caused by diving accidents \cite{4,5}. Patients with cervical injuries and drowning usually have signs of serious injury or history of high-impact trauma before submersion. We present the case of a patient with near drowning associated to a cervical spine injury with initial normal x-rays.

2. Case report

This is a 47-year-old previously healthy male tourist. He was swimming in the ocean and was rescued by other bathers because he was seen drowning in the seawater; he was taken to the shoreline and was resuscitated there. Nobody witnessed the accident; so, there were no initial details about the exact mechanism of the injury.

The patient was admitted to the hospital with oxygen saturation of 60\%, hypotension (80/40 mm Hg), dyspnea, bilateral crackles, and generalized cyanosis. Neurologically, he was stuporous, pupils were equal and reactive, and motor function on limbs was absent even with painful stimuli. Because of the critical status of the patient, cardiopulmonary resuscitation was performed and he was intubated, sedated, and transferred to intensive care unit. The evolution about the ventilatory problem was satisfactory 24 hours after the sedation was withdrawn and the tracheal tube was removed. After this, the patient was alert but was not able to move his limbs. Because of this, a consultation to neurosurgery was asked.

The patient was alert, his speech was normal, he had amnesia for the accident, and his pupils were equal and reactive and ocular movements normal. Limbs were paralyzed. Strength on both upper limbs was 0/5, left lower limb 0/5, and right lower limb 2/5. Sensation below shoulders was absent. Miothetic reflexes were exalted and extensor plantar response on left side was present.

A lateral cervical x-ray (Fig. 1) only showed degenerative changes in C6-7 level, characterized by disc space collapse and osteophytes, and no fractures or dislocations; because the cervical radiographs were normal and because of the near-drowning history and severe hypoxemia, cervical magnetic resonance imaging was performed to rule out any ischemic injury to the spinal cord or a traumatic...
injury not evident in plain films. Magnetic resonance imaging showed spinal cord compression secondary to a C3-4 herniated disc. Sagittal T1-weighted image (Fig. 2) shows a C3-4 disc herniation compressing the spinal cord; the posterior longitudinal ligament is intact. The axial views showed that the herniation was most important on the left side. Because the accident was 24 hours earlier, methylprednisolone treatment was not used.

The patient was operated on to decompress the spinal cord. An anterior approach, C3-4 discectomy, fusion, and plating were performed.

Postoperatively, the patient presented slight improvement of strength on limbs. In the following days, he continued showing improvement in strength and sensation on 4 limbs. The patient was discharged to a rehabilitation facility to continue treatment. One year after the injury, he is able to walk with a cane.

Regarding the accident, the only part that he remembered is that he was rolled over by a wave, causing him to lose strength on 4 limbs and not able to swim; he denied any prior cervical spine symptoms.

3. Discussion

Most drowning cases occur in freshwater. The pathological differences between freshwater and saltwater drowning are hypertonicity status and significant pulmonary edema, which occur more frequently in saltwater drowning; however, the final result is the same in both conditions: hypoxia [6,7].

Cervical injuries are the most common associated injuries in near-drowned or drowned patients, and usually, cervical trauma with secondary neurological deficit and inability to swim or surface is the cause of drowning or near drowning. Patients who dived in shallow waters often have an associated fracture or dislocation of cervical spine; those who have entered the water from a height may also have intra-abdominal, thoracic, and spinal injuries [8].

We reviewed the literature about spine injuries related to patients with near drowning or drowning. We found only small reports of cases and 2 series with important number of patients. In a series of children and adolescents, Hwang et al [4] found a prevalence of 4.9% (7/143) of cervical injuries related to submersion; 5 presented fractures, 1 dislocation, and 1 cord contusion. All injuries occurred in swimming pools, 6 were caused by diving, and none of these patients died. Watson et al [5] reported a review of cervical spine injuries in submersion patients and the prevalence of cervical spine injury was 0.49 % (11/2244); 10 of 11 patients died. In this series, every patient presented vertebral fractures and had history of high-impact trauma, which warned emergency physicians about the possibility of cervical spinal cord injury.

Posttraumatic disc herniation is not a common event; most of them are secondary to motor vehicle accidents and falls [9]. This kind of injury can be secondary to a hyperflexion or hyperextension mechanism [9-11].

In the present case, the patient was swimming in the ocean and was caught up in a wave and driven to the sea bottom, presenting a cervical injury due to a disc herniation. This produced paralysis in limbs and inability to swim, resulting in near drowning.

In near-drowned patients, cervical spine injuries must be suspected in patients with diving or high-impact trauma, physical evidence of severe trauma, and positive neurological and radiographic findings. Because the accident was not witnessed, there were no signs of trauma and the initial cervical x-rays were normal; none of this information was present in our patient. Moreover, the coma status secondary...
to brain hypoxia did not allow an adequate neurological examination.

To our knowledge, this is the first case reported of near drowning secondary to posttraumatic disc herniation.

4. Conclusion

Emergency department physicians should be aware about this type of cervical injury. A posttraumatic disc herniation must be ruled out in near-drowned patients who present normal cervical radiographs and have neurological findings suggestive of cervical spinal cord injury.

References


Duret hemorrhage is not always suggestive of poor prognosis: a case of acute severe hyponatremia

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1. Introduction

Delayed brainstem hemorrhage secondary to descending transtentorial herniation is known as Duret hemorrhage \cite{1}. Duret hemorrhages have mostly been reported in trauma victims with cranioencephalic lesions, including acute subdural hematoma and intraparenchymal hemorrhages \cite{1,2}. As the patients died in most cases, Duret hemorrhage has been suggestive of poor prognosis.

2. Case report

A 30-year-old man who had been admitted to a mental hospital with a diagnosis of schizophrenia was found having clonic seizures early in the morning and was admitted to the ED in a deep comatose state. Polydipsia had been observed for several days. On arrival, the Glasgow Coma Scale of the patient was 3/15 with fully dilated and nonreactive pupils. Laboratory examination showed severe serum electrolyte abnormalities: sodium concentration of 103 mEq/L, chloride concentration of 70 mEq/L, and an osmolality of 216 mosm/kg. Computed tomography (CT) of the brain showed severe diffuse cerebral edema and obliteration of the cisterns (Fig. 1). Acute severe hyponatremia resulting in serious brain swelling causing descending transtentorial herniation was diagnosed. Rapid correction of the serum sodium concentration was immediately initiated by the infusion of 10% glycerol (200 mL, 4 times a day) and hypotonic electrolyte solution. The serum sodium concentration reached 121 mEq/L during the first 12 hours and his neurological symptoms improved: he withdrew his right extremities from pain and his pupils were reactive, although asymmetric (right, 4.5 mm; left, 3.0 mm). Repeat CT showed a paramedian high-density area in the mesencephalon extending to the upper pons and low-density areas in the right posterotemporal and occipital regions despite a remarkable improvement in the brain swelling. Magnetic resonance imaging (MRI) suggested a paramedian hemorrhage in the upper brainstem and infarction in the right posterotemporal and occipital regions (Fig. 2).

The rate of correction of the serum sodium concentration was slowed to less than 10 mEq/L per day to a normal serum sodium concentration. His neurological findings ameliorated gradually: he opened his left eye in response to pain on day 6, voluntarily moved his right arm and leg on day 17, and became alert on day 23. He was discharged from the hospital on day 62 with only slight left paresis.

3. Discussion

One of the most common causes of severe hyponatremia is excessive intake of water caused by psychogenic polydipsia \cite{3}. In acute hyponatremia developing within a short period (usually 2-3 days), cerebral swelling may result, as the serum sodium concentration suddenly falls without a sufficient time to develop an adaptative response of extruding intracellular electrolytes and the consequent osmotic gradient leads to a rapid shift of water from the...
extracellular to the intracellular compartment [3,4]. Acute hyponatremia should be corrected immediately and rapidly to prevent brain swelling, and a rate of correction of 1.0 to 2.0 mEq/L per hour to a serum sodium concentration of 120 to 125 mEq/L is recommended [5]. Although the risk of neurological sequelae induced by the rapid correction is minimal [6], slow correction often results in severe neurological damage or herniation and death.

In the present case, the severe hyponatremia had acute features, and the serum sodium concentration was corrected

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**Fig. 1**  Computed tomography scan of the brain on admission showing diffuse cerebral swelling causing descending transtentorial herniation.

**Fig. 2**  Computed tomography scan (upper), diffusion-weighed MRI (middle) and flare MRI (lower) showing significant improvement in the brain swelling with Duret hemorrhage and infarctions in the parietotemporal and occipital regions.
immediately and rapidly, resulting in the improvement of brain swelling as well as clinical symptoms. However, both the repeat CT and MRI showed delayed upper brainstem hemorrhage.

Duret hemorrhage is a brainstem hemorrhage due to descending transtentorial herniation of any cause. Duret hemorrhages are located generally in the midline, paramedian, and ventral regions in the tegmentum of the upper pons and midbrain. It is believed that the pathogenesis of Duret hemorrhages is the distortion of pontine perforating branches against the relatively immobile basilar artery, due not only to a caudal displacement of the upper brainstem by descending transtentorial herniation but also to an anterior-posterior elongation of the brainstem by side-to-side compression [1,2]. The brainstem hemorrhage in the present case was secondary to the transtentorial herniation and occurred in the paramedian area, which is compatible with Duret hemorrhage.

The outcome of patients with Duret hemorrhage has been reported to be almost always fatal. However, our patient not only survived but also had no neurological sequelae, except for a slight hemiparesis. We could find only one report in which the patient survived with minimal neurological defects after descending transtentorial herniation with Duret hemorrhage caused by an extradural hematoma after craniotomy [7]. In that case, surgical decompression promptly resolved the transtentorial herniation. Perhaps rapid resolution of the transtentorial herniation after early diagnosis and treatment without delay in both cases contributed to the relatively good prognosis.

We conclude that Duret hemorrhage is not always suggestive of a poor prognosis if it is secondary to brain swelling, which is rapidly reversible by immediate treatment of the cause after early diagnosis.

References

Case Reports

Lateral abdominal wall hematoma due to tear of internal abdominal oblique muscle in a patient under warfarin therapy

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Abdominal pain is a common problem in the ED. Abdominal wall hematoma is a rare cause of acute abdominal disorders and may be misdiagnosed from other causes of abdominal pain. We present a patient who visited our ED because of acute left flank and left lower abdominal pain. In the present case, the ultrasonography (US) and computed tomography (CT) showed not only the hematoma but also the muscular defect of left internal abdominal oblique muscle which suggested the bleeder. Angiography showed active extravasation from left deep circumflex iliac artery, and \textit{N}-butyl cyanoacrylate glue embolization was performed successfully.

On arrival, a painful mass was palpable in the left lower flank. Her vital signs were as follows: blood pressure, 163/76 mm Hg; pulse, 88 beats per minute; body temp-
perature, 36.2 °C; respiratory rate, 16 breaths per minute. The laboratory findings were as follows: hematocrit, 0.32; hemoglobin, 10.4 g/dL; white blood cell count 9550/μL; and platelets, 232 000/μL. The international normalized ratio of prothrombin time was 5.3, but the partial thromboplastin time was within reference range. Abdominal US revealed an 11-cm fresh abdominal wall hematoma. Moreover, a second US on the next day showed a focal discontinuity of left internal abdominal oblique muscle (Fig. 1). The contrast-enhanced CT showed an extensive hematoma with focal fluid-fluid level over the lateral abdominal wall. An active bleeder with contrast extravasation was noted within the hematoma (Fig. 2). Ecchymosis developed on the next day, and hemoglobin dropped to 7.2 g/dL. Emergency angiography was performed, and an active extravasation from left deep circumflex iliac artery via the left femoral artery was noted (Fig. 3). Embolization with 20% N-butyl cyanoacrylate glue was successfully performed. The postembolization course was uneventful. A third US showed that the size of hematoma decreased gradually, and the patient was discharged from hospital 18 days later.

Spontaneous rectus sheath hematomas caused by the rupture of the inferior epigastric artery are well known [1]. However, hematomas of the lateral abdominal wall caused by a rupture of the deep circumflex iliac artery are very rare. Several predisposing factors are identified, such as bleeding tendency, use of anticoagulants, overcontraction or overstretching of the muscle as a result of coughing or vomiting, and weakness of vascular wall as a result of old age or arteriosclerosis [2,3].

Abdominal wall hematomas have been mistaken for common acute abdominal diseases, such as appendicitis, urinary obstructions, acute cholecystitis, incarcerated inguinal hernias, dissecting aortic aneurysms, mesenteric vascular insults, pregnancy, and torsion of an ovarian cyst [4]. The US and CT can provide useful information for the differential diagnosis [5]. In the present case, we showed not only the hematoma, but also the muscular defect of left internal abdominal oblique muscle.

Conservative treatment is acceptable for most patients, and surgical treatment is limited to some conditions, such as hematoma progression, a rupture into the peritoneal cavity, or infection. Recently, several reports have demonstrated that angiography with embolization can control bleeding and avoid surgical intervention in some cases [6,7].

For the emergency physicians, accurate differential diagnosis for acute abdominal pain is important because it can determine appropriate treatment. Although spontaneous abdominal wall hematoma is rare, it should be taken into consideration in high-risk patients. The US and CT are good modalities for initial differential diagnosis, and angiography with embolization is effective in the treatment of selected patients with abdominal wall hematoma.

References

Correspondence

Distinguishing between aortic dissection and dissecting aneurysm— precise use of new nomenclature

To the Editor,

We’ve read the case report from Linett [1] titled “Dissecting abdominal aortic aneurysm in a young man: an uncommon presentation of abdominal pain” published in the May issue of AJEM. The authors unfortunately used outdated and somewhat confusing nomenclature.

After Morgagni [2] described the pathological findings, “aortic dissection” and “dissecting aneurysm” are phrases used interchangeably in the literature and medical community. But actually, the definition of dissection is “blood enters into the media or the potential space between intima and media and creates a false lumen which then spreads in an antegrade or retrograde manner within the vessel.” It doesn’t necessarily imply aneurysmal dilatation in the affected vessel. On the other hand, true aneurysms involve dilatation of all 3 arterial wall layers. Both the definitions and pathophysiologies of dissection and aneurysm are different. Therefore, the recent review articles [3,4] and the International Classification of Diseases, Ninth Revision, Clinical Modification, in 2001 [5] all suggest that dissecting aneurysm is a misnomer and should not be used anymore. In the selected figures of the article of Linett [1], no obvious aneurysmal dilatation can be found in the dissecting segment. And no dissection can be found in the displayed aneurysm. The low-density crescent lesion is considered to be mural thrombus using the calcified intima as landmark. This also supports the nomenclature change in the International Classification of Diseases, Ninth Revision, Clinical Modification, in 2001 [5].

Precise nomenclature is critical to effective scientific communication, and this case illustrates the need to stay highly current to avoid out-of-date nomenclature and confusion.

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References


Prevalence of postdural puncture headache after ED performed lumbar puncture

To the Editor,

Lumbar puncture (LP) is a common diagnostic procedure performed by emergency medicine (EM) physicians to evaluate patients with a concerning headache or altered mentation. Bier was the first to hypothesize that this severe postural headache resulted from a persistent cerebrospinal fluid leak caused by a dural tear [1,2]. Postdural puncture headache management is difficult and often requires prolonged bed rest, hospital admission, various analgesics (caffeine, opioids), and/or an epidural blood patch [1,2].

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There are numerous factors that modify the risk of developing a PDPH. Perhaps the most important of these is needle size and design. Numerous trials have shown that the use of smaller noncutting (atraumatic) needles significantly reduces the risk of PDPH (Fig. 1) [3-5]. Review of the current literature reveals rates from 1% to 11% with noncutting needles (Whitacre, Sprotte, Gertie Marx) vs 5% to 25% with conventional cutting needles (Quincke) [4-6]. Despite these findings, emergency department (ED) diagnostic LP continues to be routinely performed using large (20-22 gauge) Quincke cutting needles. To our knowledge, there have been no studies examining the prevalence of PDPH in an ED population.

The purpose of this investigation was to determine the prevalence of PDPH in patients undergoing diagnostic LP by ED MDs. We also compared complication rates between 20- and 22-gauge needles.

We performed a prospective, multicenter, observational trial of adults (age ≥18 years) who underwent diagnostic LP in the ED from October 2002 to March 2003.

Study enrollment took place at two urban EDs with an EM training program and an approximate combined annual census of 200,000. All consenting adult patients (age ≥18 years) undergoing LP in the ED were enrolled. Patients were excluded if they were incarcerated, unable to give informed consent, or had an LP within 5 days. This trial was approved by the institutional review board governing both hospitals.

Only 20- or 22-gauge cutting (Quincke) needles were used at either site. Although patients were not randomly assigned to a needle group, each site had either needle (but not both) in their standard LP kit. Standardized data collection sheets were used to record basic demographic information, level of physician training, reason for procedure, number of attempts before successfully obtaining cerebrospinal fluid, patient positioning, and needle bevel direction relative to dural fibers.

The main outcome measure was the development of a PDPH. Secondary outcomes included effect of physician level of training, needle bevel direction, and patient positioning on development of PDPH as well as required treatment(s) for PDPH.

Study investigators contacted subjects via telephone at least once within 7 days after LP. Most patients were contacted at least twice during that period. If a complication occurred, the patient was followed up until symptoms resolved.

Telephone follow-up consisted of simple focused standardized questions regarding headache by trained investigators. Data were analyzed for overall prevalence and complication rates between both groups (20- and 22-gauge needles).

A total of 142 patients were enrolled ranging in age from 18 to 94 years (mean, 39 years). This included two protocol violations (prisoners). Forty percent of enrolled patients were men. One hundred six patients were completely followed up. Sixteen patients had a PDPH resulting in an overall prevalence of 15.1% (Fig. 2). Analysis of groups by needle size showed a significant difference in PDPH rates between 20- and 22-gauge Quincke needles (30% vs 6.1%; P = .002 Fisher exact test). Of patients with a PDPH, hospital admission for blood patching was required in 4 (33%) of 12 patients in the 20-gauge group and none in the 22-gauge group. These 4 patients also required inpatient caffeine and narcotic therapy. There were no differences in complication rates based on reason for LP, number of attempts, patient positioning, needle bevel direction, or physician level of training.

Postdural puncture headache is a debilitating complication of diagnostic LP, a procedure commonly performed by EM MDs. An understanding of the prevalence of this complication is important for each emergency physician and facilitates informed patient consent.

In this prospective observational trial, we found that the overall prevalence of PDPH is higher than reported in the anesthesia literature for smaller atraumatic needles. In addition, the use of 20-gauge needles increased the occurrence of PDPH. This group also required more interventions for symptom resolution in the form of a blood patch as compared with the group who had a PDPH with a 22-gauge needle (4 vs 0). This suggests that PDPH in this group was more severe.

Fig. 1 Graphic illustration of the cutting bevel of the commonly used Quincke needles and noncutting bevels frequently used by anesthesiologists.

Fig. 2 Summary of enrolled patients and outcomes.
The prevalence of PDPH after ED performed LP is high and seems to be related to needle size and design. Emergency medicine MDs should preferentially use smaller noncutting needles.

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References

Accuracy of weight estimation by ED personnel

To the Editor,

Emergency physicians routinely treat patients who require medications whose dosages are weight based. Because time constraints and patient immobility often preclude obtaining an actual weight during ED evaluation, the 2 most common methods for arriving at a dosage are weight estimation and dosing based on the “standard” of a 70-kg man and 60-kg woman [1]. The purpose of this study is to evaluate the accuracy of health care provider estimation and patient self-report of weight. In addition, we wish to determine whether the 70-kg standard is reasonable for our patient population.

This study was a prospective observational study conducted at Beth Israel Deaconess Medical Center ED. A convenience sample of 440 ambulatory patients was enrolled over a 6-month period. We restricted our sample to patients who were ambulatory at triage because they would have to stand on a scale. No children are seen in our ED, so our study sample was limited to an adult population (age >18 years). Institutional review board approval for the study was obtained.

Fig. 1 Scattergrams of estimated vs actual weight for patients, doctors, and nurses.

Accuracy of weight estimation by ED personnel

To the Editor,

Emergency physicians routinely treat patients who require medications whose dosages are weight based. Because time constraints and patient immobility often preclude obtaining an actual weight during ED evaluation, the 2 most common methods for arriving at a dosage are weight estimation and dosing based on the “standard” of a 70-kg man and 60-kg woman [1]. The purpose of this study is to evaluate the accuracy of health care provider estimation and patient self-report of weight. In addition, we wish to determine whether the 70-kg standard is reasonable for our patient population.

This study was a prospective observational study conducted at Beth Israel Deaconess Medical Center ED. A convenience sample of 440 ambulatory patients was enrolled over a 6-month period. We restricted our sample to patients who were ambulatory at triage because they would have to stand on a scale. No children are seen in our ED, so our study sample was limited to an adult population (age >18 years). Institutional review board approval for the study was obtained.

Fig. 1 Scattergrams of estimated vs actual weight for patients, doctors, and nurses.
The physician and nurse caring for the patient were asked to estimate the patient’s weight. The patient was also asked to report his or her own weight. The patient was then weighed on a calibrated digital scale; this was considered the gold-standard weight.

Individuals were blinded to the actual weight and to other individuals’ estimates. Measure of estimation error [absolute value of (estimate − actual)/actual] was used to determine accuracy. One-sample paired t test, nonparametric rank test, and regression analysis were used as appropriate with significance set at P < .05. All the statistical analyses were conducted using S-plus 2000 for Windows (MathSoft, Inc, Boston, MA).

The mean weight of our patients was 80.5 kg (interquartile, 65.7-92.5) with women having a mean of 75.0 kg (59.7-86.6), and men, a mean of 85.9 kg (73.9-95.3). There was no significant difference in weight when patients were stratified by age (age <65 or >65 years). The mean weight estimate by doctors was 74.5 kg (63.5-83.9) and 75.3 kg (63.5-83.9) by nurses.

Patients were the most accurate at estimating their own weight. The error of patient’s self-estimation of 3.0% (2.1%-4.1%) was significantly better than the doctor’s 11.1% (4.3%-16.3%) or nurse’s 10.9% (4.8%-16.0%) based on 1-sample paired t and nonparametric rank tests (all P < .01). Using regression analysis, the heavier the patient, the less accurate the doctor’s and nurse’s estimation (both P < .01). The estimates for the 3 different groups are represented graphically in Fig. 1. Physician and nurse estimations were not significantly different, nor was a significant difference found in weight estimation between patient sex or estimator sex.

In our study, health care providers proved to be unreliable estimators of patient weight. In 14% of the cases, the estimates were off by a magnitude of more than 20% from the actual weight. Patients proved to be significantly more accurate. Only 2.7% of patient estimates had an error of estimation of greater than 10%, and no patient estimates had an error of 20% or greater.

Other studies have looked at the accuracy of weight estimation of health care providers in various settings [2-4]. None of these studies have found health care providers to be reliable weight estimators. Our study found a tendency to overestimate the weight of patients less than 60 kg and underestimate the weight of those more than 70 kg.

The use of the 70-kg standard as a standard weight estimate for all adults also proved to be a poor tool in our population; 52% of women and 81% of men weighed more than 70 kg. Based on these results, we can neither recommend weight estimation as a reliable tool nor recommend the dosing of critical medications based on the commonly accepted 70-kg average.

Our study was limited by a potential selection bias in that we only included ambulatory adults. It is possible that patients who arrived by stretcher have different anthropometric characteristics, and their actual weight or the weight estimates may be different than those in our study population. This may be particularly true of the debilitated elderly who have been found to have a decline in body weight with aging [5]. We also cannot generalize our results to children.

The most accurate means of obtaining a weight is by direct measurement. If this is not feasible, patients should be asked their weight as this was found to be the most accurate method of weight estimation and is significantly better than nurse or physician estimation. In our patient population, the use of the 70-kg standard for delivery of medications would significantly underdose many of our patients. The clinical effect of underdosing and alternatives to the 70-kg standard need to be considered.

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References

ED environmental tobacco smoke counseling

To the Editor,

Children with asthma have a decrease in pulmonary function, an increase in airway reactivity, an increase in the frequency of ED visits for asthma [1], and an impaired recovery after hospitalization for asthma as a result of environmental tobacco smoke (ETS) exposure [2]. By providing ETS reduction

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education to parents who bring their children to the ED for smoking-related illnesses such as asthma, ED physicians can aid in accomplishing the Healthy People 2010 goal of reducing the proportion of ETS-exposed children to 10% [3], while using a potentially “teachable moment” [4].

We conducted a pilot study to investigate whether an ED educational intervention for parents of asthmatic children on ways to modify and stop smoking as a means of reducing children’s ETS exposure would result in changes in smoking habits. Eligible children were aged 3 to 16 years, had a history of asthma, and presented to the ED with wheezing, labored or trouble breathing, cough, or chest pain. Parents were eligible if they reported that they smoked 1 cigarette or more per day during the previous week. The study protocol was approved by the hospital’s institutional review board. Informed consent was obtained on all subjects. Using a random-numbers table, parents were randomized into either the (1) intervention group (IG) or, (2) control group (CG).

Baseline information included questions on demographics, parents’ current smoking habits, and smoking locations, including questions from the Fagerstrom Test for Nicotine Dependence (FTND) [5] and stage-of-change algorithm [6]. The FTND assesses an individual’s level of addiction; a score of 4 or greater is used to identify moderately to highly nicotine-dependent individuals [5], and the stage-of-change algorithm is used to determine the smoker’s readiness to quit [6]. Child baseline urine samples were collected and assayed for cotinine by gas chromatography nitrophosphorus detection with a lower limit of sensitivity of 0.2 ng/mL. Cotinine is a metabolic derivative of nicotine that is excreted in the urine and serves as an accurate and valid biochemical marker of recent and short-term ETS exposure in children with asthma [7-9]. Subjects returned to the ED for a study visit 1 month after the baseline visit during which parents completed the same baseline questions, excluding demographic differences analysis of variance. The least square means were reported.

### Table 1: Demographics of the study population and compliance with 1-month follow-up

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CG (n = 82)</th>
<th>IG (n = 83)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of parent (y)</td>
<td>30.8 ± 6.5</td>
<td>33.2 ± 9.1</td>
<td>.05</td>
</tr>
<tr>
<td>Age of child (y)</td>
<td>7.6 ± 3.1</td>
<td>8.1 ± 3.3</td>
<td>.3</td>
</tr>
<tr>
<td>No. of years smoked (y)</td>
<td>13.5 ± 7.6</td>
<td>15.8 ± 9.4</td>
<td>.08</td>
</tr>
<tr>
<td>Female parents</td>
<td>75 (91.5%)</td>
<td>71 (85.5%)</td>
<td>.2</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>26 (31.7%)</td>
<td>22 (26.5%)</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>56 (68.3%)</td>
<td>61 (73.5%)</td>
<td>.5</td>
</tr>
<tr>
<td>Employed</td>
<td>52 (63.4%)</td>
<td>48 (57.8%)</td>
<td>.5</td>
</tr>
<tr>
<td>Baseline urine cotinine</td>
<td>62 (75.6%)</td>
<td>60 (72.3%)</td>
<td>.6</td>
</tr>
<tr>
<td>Completed 1-mo survey</td>
<td>41 (50%)</td>
<td>41 (49.4%)</td>
<td>.9</td>
</tr>
<tr>
<td>1-mo Urine cotinine</td>
<td>36 (43.9%)</td>
<td>34 (41%)</td>
<td>.7</td>
</tr>
</tbody>
</table>

Analyses were conducted using the Statistical Package for Social Sciences (Chicago, IL) version 11.5. Categorical data were analyzed using χ² and Fisher exact tests, and continuous data were analyzed using t tests and repeated-measures analysis of variance. Because the distribution of the urine cotinine values was highly skewed to the right (skewness, 1.9), the log-transformed cotinine values were analyzed. We first compared the 2 groups on their individual baseline and 1-month values, and we then calculated the difference scores for subjects who remained in the study at 1 month from their baseline values using the Student t test and the nonparametric Wilcoxon rank sum test. Potential confounders, including sex, and parental socioeconomic status were considered using analysis of variance. The least squared means were reported.

### Table 2: Comparison of smoking characteristics between groups at baseline and at 1 month

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CG (n = 82)</th>
<th>IG (n = 83)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>FTND</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 165)</td>
<td>3.72 ± 1.5</td>
<td>3.84 ± 1.3</td>
<td>.6</td>
</tr>
<tr>
<td>1 mo (n = 82)</td>
<td>4.05 ± 1.4</td>
<td>3.29 ± 1.4</td>
<td>.02</td>
</tr>
<tr>
<td>Stage of change: precontemplation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 165)</td>
<td>28 (34.1%)</td>
<td>33 (39.8%)</td>
<td>.6</td>
</tr>
<tr>
<td>1 mo (n = 82)</td>
<td>8 (19.5%)</td>
<td>2 (5.1%)</td>
<td>.05</td>
</tr>
<tr>
<td>Smoked outside only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 155)</td>
<td>14 (18.4%)</td>
<td>21 (26.6%)</td>
<td>.2</td>
</tr>
<tr>
<td>1 mo (n = 80)</td>
<td>13 (31.7%)</td>
<td>23 (59%)</td>
<td>.01</td>
</tr>
<tr>
<td>Smoked in 1 room only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 141)</td>
<td>14 (20%)</td>
<td>9 (12.7%)</td>
<td>.2</td>
</tr>
<tr>
<td>1 mo (n = 79)</td>
<td>10 (24.4%)</td>
<td>7 (18.4%)</td>
<td>.5</td>
</tr>
<tr>
<td>Informed consent</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 142)</td>
<td>6 (8.5%)</td>
<td>5 (7.0%)</td>
<td>.8</td>
</tr>
<tr>
<td>1 mo (n = 80)</td>
<td>4 (9.8%)</td>
<td>2 (5.1%)</td>
<td>.4</td>
</tr>
<tr>
<td>Open windows while smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 145)</td>
<td>25 (35.2%)</td>
<td>20 (27%)</td>
<td>.3</td>
</tr>
<tr>
<td>1 mo (n = 80)</td>
<td>16 (39%)</td>
<td>10 (25.6%)</td>
<td>.2</td>
</tr>
<tr>
<td>Changed clothes after smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline (n = 140)</td>
<td>1 (1.4%)</td>
<td>0 (0%)</td>
<td>.5</td>
</tr>
<tr>
<td>1 mo (n = 80)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0</td>
</tr>
</tbody>
</table>

### Notes

- Behavior reported as occurring “most times or always.”

with ways to decrease their child’s ETS exposure based on their living conditions. Smoking cessation advice consisted of recommendations adapted from the Clinical Practice Guideline: Treating Tobacco Use and Dependence [10]. Both parents in the IG and CG were given pamphlets from the American Cancer Society with smoking cessation information; CG parents were not given specific ETS information.
We recruited 165 parent/child subjects. There were no statistically significant differences between the 2 groups (IG, n = 82; CG, n = 78) on any baseline variable, and there were no statistically significant differences between subjects who did and did not complete follow-up on any baseline variable (Table 1). Fifty percent (n = 82) of subjects were contacted at 1-month follow-up. Subjects lost to follow-up were considered continuing smokers, using the “intent-to-treat” model of analysis. Table 2 shows the following 1-month differences between the IG and CG subjects: smoked outside only (59% and 31.7%, P = .014) and the FTND (3.3 ± 1.4 and 4.1 ± 1.4, P = .02). Although not statistically significant, 4 (0.05%) subjects (CG, 1; IG, 3) reported quitting smoking at 1 month. There were no significant differences in baseline cotinine levels between the CG and the IG groups (mean ± SD, 31.71 ± 42.7 vs 25.15 ± 29.77, P = .8), but there was a 22% decrease in cotinine levels in children of IG at 1 month compared with baseline (P = .1) and a 39% increase in cotinine levels in children of CG (P = .05).

This pilot study demonstrates encouraging trends toward a change in parental smoking habits after a brief ED intervention. Additional research is needed to determine ways to improve long-term study compliance and follow-up and appropriate baseline and booster sessions about ETS reduction techniques in an ED population.

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References


Discordant results in x-ray interpretations between ED physicians and radiologists. A prospective investigation of 30,000 trauma patients

To the Editor,

Errors in radiological interpretation by nonradiologists continue to have a negative impact on the quality of care for ambulatory trauma patients in the emergency department (ED). Numerous studies demonstrate that radiologists are more capable of accurate interpretation of x-rays than physicians who are not certified in this specialty [1-10]. Emergency department patients sustaining misdiagnosed fractures run the risk of diminished quality of care by treatment based on inaccurate radiological assessment. Caregivers are at heightened risk for medicolegal liability stemming from diagnostic error.

We report a prospective study based on 30,000 patients who had radiographic assessment for trauma and who visited the ED of a university hospital and were not admitted (from 1999 to 2000). The goal is to evaluate the role of specialized double reexamination in the prevention of radiological misinterpretation and in the reduction of inappropriate therapeutic interventions arising from these errors.

All patients received standard radiographs (front and lateral view) for their respective types of trauma. The study included patients of both sexes and all ages. Subjects presented no sex ratio with ages ranging from 15 months to 94 years and a median age of 25 years. The radiographs were read first by a nonradiologist ED physician. A second review of the patient’s x-rays was performed by an ED physician as early as the day after their visit to the hospital. A third review of the patient’s x-rays was then performed by a radiologist 24 hours after the second reading. All reexamined radiographs were analyzed in conjunction with an account of the presenting clinical signs and symptoms.

We studied the following items: type of traumatic pathology, age of patients, and misinterpreted cases requiring change in treatment.

Patients whose radiological interpretations were found to be in error were recalled to the ED.

1. Discordant results in 312 (1.04%) cases detected during the second analysis by an independent ED physician.
2. Discordant results in 416 (1.38%) patients disclosed by a radiologist after the second reading done by the emergency physician. One hundred four additional cases were found to have discordance by this third review.

Of the 30,000 cases reexamined, 413 (1.37%) revealed fractures and 3 (0.01%) revealed other lesions, as Table 1 shows.

Of the patients presenting with errors in radiological interpretation, 240 (57.7%) involved patients between 6 and 15 years of age. In 97 (23.32% of all radiographs erroneously interpreted), inadequate treatment resulted from erroneous interpretation of the images. This represents 0.32% of the original 30,000 cases.

Our study suggests that a system of routine reexamination, particularly when coupled to heightened awareness and skill in the analysis of radiographs most likely to be misinterpreted, can be an effective solution. Indeed, systematic reexamination of archived radiographs by radiologists is among the approaches recently proposed by the Agency for Healthcare Research and Quality (Picture Archiving and Communication System) [11]. Nevertheless, not every hospital is equipped with Picture Archiving and Communication System. Therefore, specialized training for nonradiologist ED physicians remains essential [11].

Our finding calls for further comment because 104 additional cases (0.35% of the 30,000 original cases but a full 25% of the cases reread by the radiologist) were found to have errors in x-ray interpretation after they had been reviewed on two previous occasions by nonradiologist physicians. Our investigation demonstrates that of 30,000 radiographs interpreted by physicians who were not radiologists, 104 (0.35%) were subject to discordant interpretation. A recent study by Espinosa and Nolan [12] showed that reexamination of x-rays caused the rate of errors in interpretation to fall significantly and the level of patient care to rise. The rate of false-negative errors fell to 0.3%. In our study, the rate of false-negative errors is about 1.3%. We have never encountered a negative reaction by patients who have been recalled because of an error in x-ray interpretation.

A study performed by Brunswick et al [13] in a university pediatric ED revealed that the error most frequently made by interns concerned x-rays of injuries to children’s limbs (23%). In our study, performed in a university adult and pediatric ED, 58% involved epiphyseal fractures. In our study, 10% of the misinterpreted radiographs in children led to a change in management. A survey of the literature on this subject corroborates our findings [8,14]. It is also important to remember that a radiological interpretation is not a de facto criterion standard. Radiologists only agree about 95% of the time [15], showing variation between experienced observers in the interpretation of accident and emergency radiographs. In our study, 97.8% of the ED radiographs were read correctly on initial interpretation by ED attending physicians; Brunswick et al [13] reported a range of 99%. In our opinion, this investment of time in specialized double reexamination has a direct bearing on the quality of patient care.

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[4] Lutkin KC, Smith SW, Matticks CA, Brunette DD. Radiologists’ review of radiographs interpreted confidently by emergency physicians

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Categorization of errors according to pathologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesions</td>
<td>Cases (N = 416)</td>
</tr>
<tr>
<td>Fractures</td>
<td>413</td>
</tr>
<tr>
<td>Phalanges (fingers and toes)</td>
<td>145 (35.1)</td>
</tr>
<tr>
<td>Epiphyseal (Salter-Harris I or II)</td>
<td>242 (58.5)</td>
</tr>
<tr>
<td>Cervical vertebra (C6 nondisplaced)</td>
<td>1 (0.24)</td>
</tr>
<tr>
<td>First metacarpal bone</td>
<td>3 (0.72)</td>
</tr>
<tr>
<td>Tarsal bones</td>
<td>3 (0.72)</td>
</tr>
<tr>
<td>Carpal bones</td>
<td>6 (1.44)</td>
</tr>
<tr>
<td>Humerus head</td>
<td>3 (0.72)</td>
</tr>
<tr>
<td>Lateral malleolus</td>
<td>10 (2.40)</td>
</tr>
<tr>
<td>Other lesions</td>
<td>3</td>
</tr>
<tr>
<td>Posterior dislocation of the shoulder</td>
<td>1 (0.24)</td>
</tr>
<tr>
<td>Mallet finger</td>
<td>1 (0.24)</td>
</tr>
<tr>
<td>Toe distal dislocation</td>
<td>1 (0.24)</td>
</tr>
</tbody>
</table>

Values are expressed as number (%).


Accuracy of laceration length estimation

To the Editor,

Lacerations are a frequent complaint of patients presenting to the ED. Of 110,115,000 ED visits in 2002, approximately 6.5 million (5.9%) were for injuries categorized as “open wounds.” It is estimated that 644 million dollars are paid on these injuries annually [1]. The Center for Medicare and Medicaid Services and Current Procedural Terminology billing regulations require physicians to document the location of the laceration and its length to properly bill for laceration repair [2,3].

We performed a study to determine how accurately emergency physicians estimate the length of simulated lacerations and to evaluate what aspects of a laceration might contribute to misestimates of length. A prospective, multicenter, observational survey was sent to emergency providers. The survey tool consisted of 30 two-dimensional line drawings representing lacerations of 10 different lengths in centimeters (ie, 0.5, 1.5, 2.0, 3.0, 4.5, 5.5, 7.0, 8.0, 12, and 13) and 3 shapes of increasing complexity (ie, linear, curved, and stellate). The survey was administered to all full-time emergency physicians at 5 EDs and all emergency medicine residents at one of the institutions. The following institutions were included: Baystate Medical Center, Faulkner Medical Center, New England Medical Center, Newton-Wellesley Medical Center, and St. Elizabeth’s Medical Center. No measuring devices were allowed during the survey. Each survey was completed independently. The study was approved by the institutional review board at Baystate Medical Center. Data were analyzed using descriptive statistics.

Absolute differences from actual simulated laceration length were determined for each laceration length. Surveys were sent to 85 emergency providers (54 attending physicians and 31 residents) with a 95% response rate. Thirty-six attending physicians (44%) and 17 residents (55%) reported that they never use a measuring tool to estimate the length of a laceration. Fig. 1 shows the absolute differences from the actual lengths for each laceration length and shape. Estimating errors increased with increasing simulated laceration length and complexity of shape. More than half of the surveyed emergency physicians do not use a measuring tool to estimate laceration length. When they do not use a measuring tool, they frequently misestimate the length of simulated lacerations.

Current Procedural Terminology was founded in 1966 and appears to have adopted the billing guidelines used by California Blue Cross & Blue Shield at that time. Using PubMed and Google, we were unable to find any evidence that the basic schema for billing lacerations has been studied or changed in the last 30 years [4-6]. Lacerations are billed depending on the type of repair (ie, simple, intermediate, or complex), by location, and by length [3]. Body location grouping is dependent on complexity of repair. Laceration length is the sum of all laceration lengths within a given grouped body location and repair type. Simple and intermediate repairs of facial lacerations are divided into less than 2.5 cm, 2.6 to 5.0 cm, 5.1 to 7.5 cm, 7.6 to 12.5 cm, 12.6 to 20 cm, 20.1 to 30 cm, and more than 30 cm. Simple and intermediate lacerations of other body locations are grouped

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Fig. 1 Absolute error of estimated length by simulated laceration length and shape.
into similar billing lengths, except that lacerations between 2.6 and 7.5 cm are grouped into a single billing level [3]. The impact of misestimates of laceration length on billing depends on the location of the laceration and where the actual laceration length is within the billing range. Misestimates of laceration length when the actual laceration length is close to either end of the billing range are more likely to result in incorrect billing. For example, a laceration requiring simple repair on the trunk that is 4.5 cm and is misestimated by 1 cm either too long or too short will still be within the correct billing range of 2.5 to 7.5 cm. A 4.5-cm laceration on the face that is overestimated by 1 cm will result in overbilling, but underestimates of 1 cm will not impact billing.

This study is limited in several important ways. It is a survey of simulated laceration instead of actual lacerations. Participants were asked to not use a measuring tool even if they usually do in clinical practice. The survey did not include lacerations of all shapes and sizes that normally occur in practice. Finally, the survey was not designed to determine the actual impact of misestimates of laceration length on billing.

We conclude that emergency physicians frequently misestimate the length of simulated lacerations when not using a measuring device. Misestimates increase with laceration length and complexity of shape. Misestimates of laceration length may affect patient billing. Use of a simple tape measure may improve accuracy of estimating laceration length.

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References

Nasal intubation or rapid sequence intubation

To the Editor,

Nasal intubation as a form of emergency airway management has been largely replaced by rapid sequence intubation (RSI) [1]. The benefit of RSI, by virtue of paralysis by neuromuscular blocking agents, is that it facilitates laryngoscopy. The downside is that, with paralysis, the patient becomes entirely dependent on the health care providers for oxygenation and ventilation. In nasal intubation, on the other hand, spontaneous breathing is maintained. This mitigates the harm caused by lack of adequate oxygenation and ventilation that may occur if there are difficulties in intubating the patient, or if there is an esophageal intubation that is unrecognized.

In Jackson County Hospital v Aldrich[2], an adult man was severely burned when he used a cutting torch on a barrel containing flammable liquid. After an explosion, he was engulfed in flames for 30 seconds and had extensive and deep burns including the face and neck. While en route to the hospital by ambulance, he received high-flow oxygen. In the ED, he was found to have an increased respiratory rate and had decreased oxygen saturations. The ED physician called a certified registered nurse anesthetist (CRNA) to intubate the patient. In assessing the patient by performing a “quick-look” laryngoscopy, the CRNA was unable to visualize his vocal cords. Given this, and because the patient was conscious and breathing spontaneously, the CRNA recommended nasal intubation. The ED physician, however, overruled nasal intubation in favor of RSI. During intubation, the CRNA was unable to visualize the vocal cords (she attributed this to burns, swelling, and the patient’s short mandible), and so she was not certain if the endotracheal (ET) tube was in the trachea or in the esophagus. A device attached to the ET tube did not detect the production of carbon dioxide, and the patient’s oxygen saturation did not increase. Concerns about proper placement were alleviated by the ED physician’s auscultation of breath sounds over the lung fields and the absence of sounds over the stomach. The patient then received a narcotic pain medication, which was followed within minutes by the loss of his pulse. Resuscitation efforts were unsuccessful. On autopsy, the medical examiner found the ET tube in the esophagus.

Nasal intubation is indicated when both of 2 criteria are present. The first is the inability of the health care provider to perform oral intubation using the preferred technique of laryngoscopy, for example, because the patient is either awake or is unconscious but maintains airway reflexes (the gag reflex or clenching of the jaw) that preclude adequate visualization of airway structures. The second criteria has 2 components: either the lack of availability of use of neuromuscular blockade (the prehospital setting) or the lack of reasonable assurance that laryngoscopy, even with the
muscle paralysis caused by neuromuscular blockade, will provide adequate visualization of airway structures (a known difficult airway).

The patient in Jackson County, having been severely burned, was hypoxic and, because the burns involved the face, required protection against mechanical obstruction of the airway. The patient was awake and breathing spontaneously. The CRNA, by “quick-look” laryngoscopy, recognized the presence of a difficult airway. Despite her preference for awake nasal intubation, RSI was performed on the recommendation of the ED physician. Subsequent complications resulted in the patient’s death.

Putting aside the critical importance in ensuring proper placement of the ET tube in the trachea, the degree of sophistication required in choosing between various methods of airway management is highlighted by this case. Although RSI offers important advantages over other forms of airway management, primarily, in its improving laryngoscopic view, it is not universally available (eg, to emergency medical service providers), and its use does not guarantee success (as demonstrated in this case). Consequently, despite the widespread use of the RSI procedure, nasal intubation should remain an option for health care providers in the management of ED patients.

The legal conclusion to the case was unfortunate for the CRNA [3]. The emergency physician, employed by the hospital, was protected under the state (Florida) Good Samaritan Act. The CRNA, as an independent contractor, was not protected by the Act and was held to a mere negligence standard. The jury found that the physician, by failing to ensure appropriate ET tube placement, acted with reckless disregard, thus exempting him from the Act. However, an appellate court reversed these findings and found the physician immune from liability under the Act. Accordingly, the corporation employing the CRNA was wholly liable for the damages. This result was despite the reasoning (as articulated in a dissenting opinion) that it is “illogical to infer the legislature intended application of a different standard to medical professionals working as part of the same team, affording greater protection to the person in charge than to those carrying out the orders…”[4]

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Predictors of inner-city recurrent violence-related injuries

Violence-related injury (VRI) is a major source of morbidity and mortality in inner-city populations. Nationwide, there are roughly 1.6 million ED visits for assault-related injuries annually [1]. Violence is the second leading cause of death nationally for individuals between the ages of 10 and 24 years and the leading cause of death of African Americans in this age group. In Brooklyn/Kings County, the focal area for this study, there were 189.64 annual hospitalizations per 100,000 because of violent injury [2]. This number is higher than the average VRI hospital admissions rate for New York City (156.1) [3] and the United States (63.9) [3].

ED-based research on violence prevention typically centers on victims of violence and how best to decrease the risk for recurrent VRI (RVRI) in these populations [4,5]. The approach seems valid, as individuals presenting to the ED with VRI have an increased risk of repeat injury, criminal prosecution, and 5-year mortality [6,7]. However,

<table>
<thead>
<tr>
<th>Table 1 Comparison of the demographic and social characteristics of patients with RVRI to those without RVRI</th>
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<tbody>
<tr>
<td>RVRI</td>
</tr>
<tr>
<td>Mean age ± SD (y)</td>
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<tr>
<td>Male sex (%)</td>
</tr>
<tr>
<td>Ethnicity</td>
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<tr>
<td>(African American, %)</td>
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<tr>
<td>Marital status</td>
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<tr>
<td>(single, %)</td>
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<tr>
<td>Education (less than high school diploma, %)</td>
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<tr>
<td>Childhood (single parent, %)</td>
</tr>
<tr>
<td>Employment (unemployed, %)</td>
</tr>
<tr>
<td>Mechanism (blunt, %)</td>
</tr>
<tr>
<td>Location of injury (own neighborhood, %)</td>
</tr>
<tr>
<td>Weapon of assault (gun, %)</td>
</tr>
<tr>
<td>Perpetrator (known, %)</td>
</tr>
<tr>
<td>Drug abuse (alcohol and/or drugs, %)</td>
</tr>
<tr>
<td>History of criminal arrest/conviction (%)</td>
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<tr>
<td>History of psychiatric disorder (%)</td>
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^a Student t test for age, Fisher exact test for all other variables; level of significance, P < .05.
few studies have been able to produce a quantifiable reduction in VRI through ED-based interventions [8].

This pilot study involved conducting a survey of patients who presented to Kings County Hospital Center with VRI. The design of this multidisciplinary project is the result of a collaborative effort among the Department of Emergency Medicine, Department of Preventive Medicine, and the Masters of Public Health program at SUNY Downstate Medical Center. This survey was designed to evaluate the demographic, social, and behavioral characteristics of VRI in central Brooklyn to identify the determinants with the potential to predict the likelihood of the outcome measure, RVRI. The study was approved by the institutional review boards of SUNY Downstate Medical Center and Kings County Hospital Center. The results of this study will be used to design an ED-based violence prevention program.

Patients aged between 13 and 60 years who present to the ED with VRI were enrolled in the study. Patients older than 60 years were excluded. Patients with change of mental status and victims of sexual assault were also excluded. The survey included demographic information, level of education, marital status, family unit during upbringing, place of injury, mechanism of assault, weapon of assault, history of drug/alcohol abuse, criminal arrest/conviction, previous VRI, and psychiatric disorders. Trained data abstractors administered the survey.

The outcome was a dichotomous variable for RVRI. VRIs are those injuries assessed as deliberately caused by another person. These injuries may result from assaults, fights, and family violence or abuse. RVRI was defined as occurrence of VRI in the past. A multivariable logistic regression model was generated to identify the variables that related to the dichotomous outcome of RVRI. Variables were selected based on univariate association with VRI and the statistical contribution of each variable to the model.

A total of 105 patients were enrolled (mean age ± SD, 29 ± 11 years; range, 15-60; 83% were men). Twenty-eight percent (n = 29) of patients reported prior VRI (RVRI). Characteristics of the study groups (with and without RVRI) are summarized in Table 1. Univariate analysis detected significant differences between the 2 groups only in employment status (P = .004) and history of prior criminal arrest/conviction (P = .001). The logistic regression analysis revealed that risk for RVRI increases by unemployment (odds ratio, 5.5; 95% confidence interval, 1.7-18.4, P = .004) and by history of arrest/conviction (odds ratio, 8.2; 95% confidence interval, 2.4-28.3, P = .001).

The relationship between history of criminal arrest/conviction and VRI is in agreement with the study performed by Rivara et al [6] in South Wales. According to this study, assault patients were more likely to be formally warned or convicted than other injured patients [6]. Conversely, our results differ from a case-control study conducted in New Haven by Moscovitz et al [9]. This study did not show higher rate of criminal conviction victims of VRI. However, there was a higher rate of criminal activity in their control group compared with their general population.

A strong relationship between low socioeconomic status and violence has been shown in previous studies [4]. Our study expands upon this idea by relating violence with a specific predictor related to poverty: unemployment. We showed that an unemployed state significantly increases the risk of RVRI. As more than 50% of all Central Brooklyn residents are unemployed [10], our results are very significant to understanding how one aspect of poverty relates to RVRI. Previous studies have predominately studied overall predictors of RVRI such as criminal activity, reinjury, and death, but none have addressed specific predictors such as unemployment.

The demographic characteristics of our patients were not correlated with increased risk of RVRI. For example, both of our study groups consisted of predominately young African-American men. This is consistent with previous studies which showed that most victims of violence are African-American men [7,9].

More detailed surveys and larger scale studies are needed to validate these results and help designing an ED-based violence prevention program.

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References
ANNOUNCEMENT

2006 CERTIFYING EXAMINATION IN
PEDIATRIC EMERGENCY MEDICINE

Examination Date: November 16, 2006.
Registration for re-registrants: March 16, 2006, through June 16, 2006.

The final month of each registration requires payment of a late fee.

All applicants must complete applications online during the registration periods. The requirements for online applications are found on the ABP Web site: www.abp.org. Additional information including eligibility requirements is found on the ABP Web site. Each application will be considered individually and must be acceptable to the ABP.

*You must apply to the Board through which you carry your primary certification. If you are applying through the ABEM for the 2006 Pediatric Emergency Medicine Certifying Examination, you must contact that Board for registration dates.

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