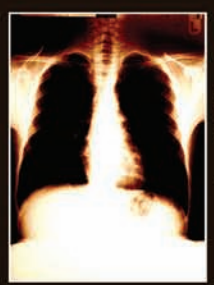
American Journal of Clinical Medicine®

Owned and Published by the American Association of Physician Specialists, Inc.

Winter 2009 • Volume Six, Number One

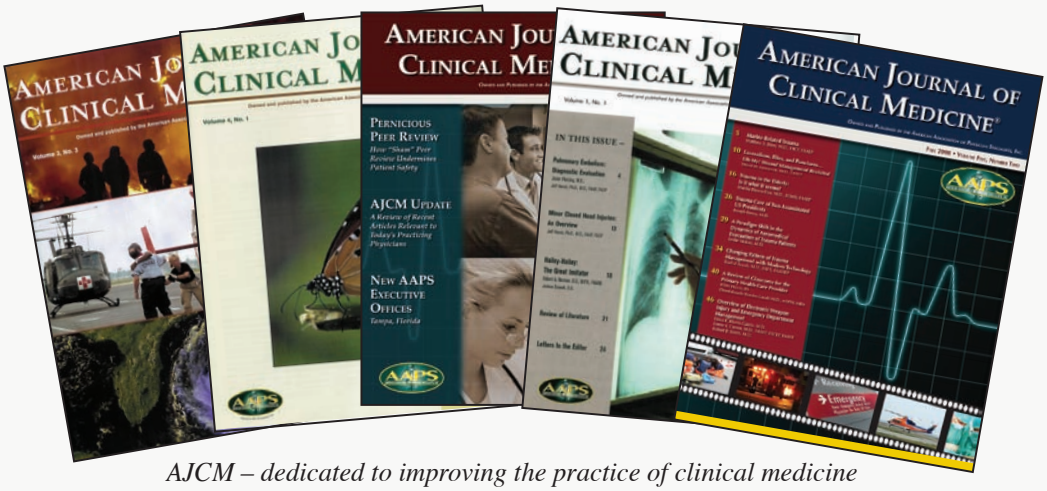
IN THIS ISSUE -



CALL FOR PAPERS

AMERICAN JOURNAL OF CLINICAL MEDICINE®

OWNED AND PUBLISHED BY THE AMERICAN ASSOCIATION OF PHYSICIAN SPECIALISTS, INC.



AJCM – dedicated to improving the practice of clinical medicine by providing up-to-date information for today's practitioners.

AMERICAN JOURNAL OF CLINICAL MEDICINE®

- **No subscription fees**
- **No physician author charges**
- **Inquiries to eberg@aapsus.org**
- **Interested physicians may submit manuscripts to editor@aapsus.org** ***(See Manuscript Criteria and Information on pages 34-35)***

The *American Journal of Clinical Medicine* (AJCM) is the official, peer-reviewed journal of the American Association of Physician Specialists, Inc. (AAPS), an organization dedicated to promoting the highest intellectual, moral, and ethical standards of its members. Its diversity incorporates physicians that represent a broad spectrum of specialties including anesthesiology, dermatology, diagnostic radiology, disaster medicine, emergency medicine, family medicine/OB, family practice, geriatric medicine, hospital medicine, internal medicine, obstetrics and gynecology, ophthalmology, orthopedic surgery, plastic and reconstructive surgery, psychiatry, radiation oncology, and general surgery.

To further the goals of AAPS, which include providing education for its members and promoting the study, research, and improvement of its various specialties, the AJCM invites submissions of high-quality review articles, clinical reports, case reports, or original research on any topic which has potential to impact the daily practice of medicine.

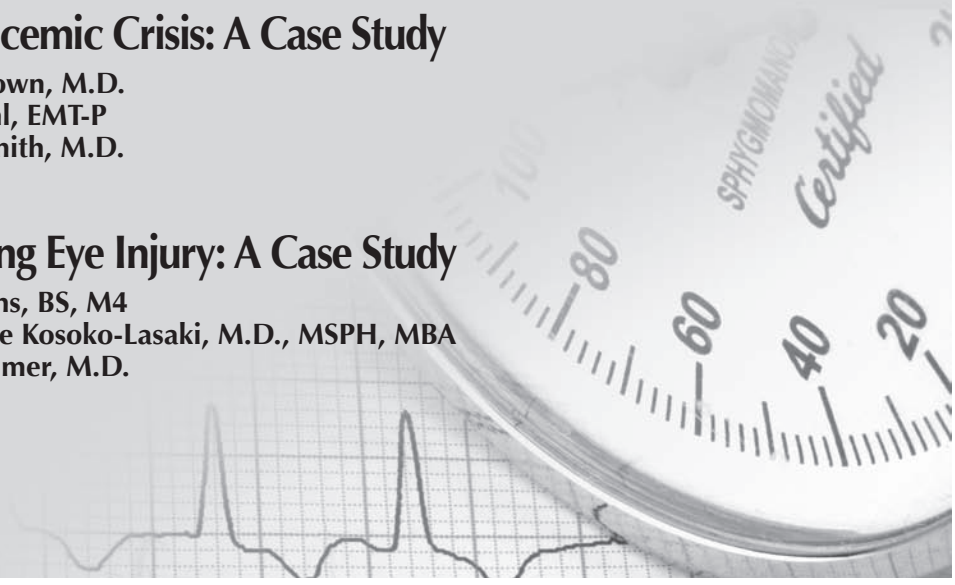
Publication in the AJCM is one of the criteria to qualify for the prestigious Degree of Fellow within the Academies of Medicine of the AAPS.

DEADLINES TO RECEIVE ARTICLES

ISSUE	DEADLINE
Summer 2009	**May 4, 2009**
Fall 2009	**August 3, 2009**

American Journal of Clinical Medicine®

WINTER 2009 • VOLUME SIX, NUMBER ONE



American Journal of Clinical Medicine®

Welcome to the *American Journal of Clinical Medicine* (AJCM) Winter 2009. The Journal is dedicated to improving the practice of clinical medicine by providing up-to-date information for today's practitioners.

The AJCM is the official journal of the American Association of Physician Specialists, Inc. (AAPS), an organization dedicated to promoting the highest intellectual, moral, and ethical standards of its members, and whose diversity incorporates physicians that represent a broad spectrum of specialties including anesthesiology, dermatology, diagnostic radiology, disaster medicine, emergency medicine, family medicine obstetrics, family practice, geriatric medicine, hospital medicine, internal medicine, obstetrics and gynecology, ophthalmology, orthopedic surgery, plastic and reconstructive surgery, psychiatry, radiation oncology, and general surgery.

Part of the mission of the AAPS is to provide education for its members and to promote study, research, and improvement of its various specialties. In order to further these goals, the AJCM invites submissions of high-quality review articles, clinical reports, case reports, or original research on any topic that has potential to impact the daily practice of medicine. Publication of a peer-reviewed article in the AJCM is one of the criteria needed to qualify for the prestigious Degree of Fellow in the Academies of Medicine.

Articles that appear in the AJCM are peer reviewed by members with expertise in their respective specialties. Manuscripts submitted for publication should follow the guidelines in The International Committee of Medical Journal Editors: "Uniform requirements for manuscripts submitted to biomedical journals" (JAMA, 1997; 277:927-934). Studies involving human subjects must adhere to the ethical principals of the Declaration of Helsinki, developed by the World Medical Association. By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of their article that might create any potential conflict of interest. More detailed information is included in the AJCM Manuscript Criteria and Information on pages 34 and 35.

In this issue we are proud to introduce a new, regular feature entitled "Medical Ethics Without the Rhetoric." Dr. Mark Pastin will offer real-life cases that present ethical issues, which are common to the practice of medicine. After reading the item, email your input to him. The next issue will feature reader perspectives and Dr. Pastin's own viewpoint. See "Medical Ethics Without the Rhetoric" on page 18.

This issue also features several articles and case studies as well as the "Sounding Board," an open forum where we offer you the opportunity to write in and express your thoughts on a subject or suggest new topics for future Sounding Boards. Letters to the editor, commenting on published articles or offering general comments or opinions, are also welcome.

This Journal represents you–we welcome your input, your articles, essays, observations, poetry, and sound bites.

The AJCM Editorial Board

American Association of Physician Specialists, Inc.
5550 West Executive Drive • Suite 400 • Tampa, Florida 33609-1035
Phone: 813-433-2277 • Fax: 813-830-6599
www.aapsus.org

Advertising: For Advertising Opportunities Contact Esther Berg or Keely Clarke at 813-433-2277.

Controversies in Prehospital Care

David M. Lemonick, M.D., FAAEP

Abstract

Nowhere in emergency medicine are mythology, legend, and tradition as conspicuous as they are in the field of prehospital care (EMS). Images of speeding ambulances with lights and sirens, aeromedical helicopters in flight, and heroic medical interventions in austere environments are awe-inspiring, thrilling, and reassuring to many of us. As dispassionate scientific scrutiny is applied to these and other practices in EMS, however, it becomes evident that many of the current practices and protocols in EMS are not based on any level of scientific evidence. This article will review current evidence about the costs and benefits of some of the most common current practices in EMS. These include the use of lights and sirens and helicopters, endotracheal intubation and its alternatives in airway management, cardiopulmonary resuscitation, advanced cardiac life support, public access defibrillation, and analgesics. It is hoped that by shining the light of scientific scrutiny upon these practices, dogma will be replaced by clinical evidence. Only in this way may cost-effective emergency care be provided for the greatest benefit to the largest number of citizens.

"It is possible to document exactly how much scientific support there is for the efficacy of our present scope of EMS practice, and it is impressively deficient."[1]

Introduction

Nowhere in emergency medicine are mythology, legend, and tradition as conspicuous as they are in the field of prehospital care (EMS). Images of speeding ambulances with lights and sirens, aeromedical helicopters in flight, and heroic medical interventions in austere environments are awe-inspiring, thrilling, and reassuring to many of us. As dispassionate scientific scrutiny is applied to these and other practices in EMS, however, it becomes evident that many of the current practices and protocols in EMS are not based on any level of scientific evidence. This article will review current evidence about the costs and benefits of some of the most common current practices in EMS. These include the use of lights and sirens and helicopters, endotracheal intubation and its alternatives in airway management, cardiopulmonary resuscitation, advanced cardiac life support, public access defibrillation, and analgesics.

It is noteworthy that, of 5,842 publications on prehospital care, only 54 were randomized controlled trials (RCTs). Of these 54 RCTs, four (7%) reported harm from the new therapy, and 74% reported no effect at all. Only seven studies (13%) of the RCTs showing a positive outcome of an intervention were not contradicted, and only one of these examined a major outcome such as survival, and only one of these was placebo-controlled.[1] Thus, there is a dearth of sound scientific support for EMS interventions, and a serious reexamination of EMS practices is needed.

It is hoped that by shining the light of scientific scrutiny upon these practices, dogma will be replaced by clinical evidence. Only in this way may cost-effective emergency care be provided for the greatest benefit to the largest number of citizens.

The Use of Helicopters in EMS (HEMS)

Medical helicopters in EMS were introduced into civilian use in the United States in 1972, and since that time there has been an exponential proliferation in their use.[2] In 2004, there were approximately 700 HEMS helicopters in the U.S., and they transported more than 300,000 patients. Last year, an estimated 400,000 people flew on EMS helicopters and the national fleet, mostly in for-profit operation, is now over 900. Thirty percent of HEMS flights are scene calls, and 70% are interfacility transports.[3] Much of the impetus for the initiation and growth of HEMS was based on the concept of a "golden hour" after trauma, popularized by Dr. R. Adams Cowley. Further, experience with combat casualties in the Korean and Vietnam wars supported the efficacy of rapid transport of wounded soldiers by helicopter. It should be noted that the very existence of such a golden hour has become the subject of debate.[4] The literature on HEMS in civilian use has been mixed though, with some critics referring to these aircraft as little more than "flying billboards."[2]

In one study comparing 337 patients transported by HEMS with 446 matched patients transported by ground ambulance, survival rates were the same. The authors concluded that there was no evidence that HEMS improved survival.[4] In another study, of 947 consecutive trauma patients transported by HEMS to Santa Clara Valley Medical Center in California between 1990 and

2000, 45% were felt to have arrived as slowly or slower than by ground EMS, and 35% of the HEMS patients were discharged directly from the ED. The authors concluded that <1% of the patients in this series had actually benefited from HEMS, and that 0.5% of the patients may have been harmed by HEMS.[5] In a Boston study of more than 1,500 trauma patients transported by HEMS from the scene, 24% were considered to have been inappropriate.[6] A metanalysis of 22 papers with a cohort of more than 37,000 trauma patients transported by HEMS found that approximately 60% were felt to have had minor injuries, and 24% were discharged within 24 hours.[7] Another study of 3,048 trauma patients transported by HEMS found that these patients had longer transport times, with no difference in mortality.[8] A 2001 study found no difference in quality of life 15 months after trauma for patients transported by HEMS compared to patients transported by ground ambulance.[9] In Houston, 122 consecutive, non-cranial penetrating trauma patients were transported by HEMS. The authors concluded that HEMS did not hasten arrival at the hospital and that scene flights for penetrating trauma in Houston were not efficacious.[10] In a 2003 review by Thomas et al. HEMS transport seemed to benefit certain patients in particular systems, while not in others.[11] One US multi-center study of blunt trauma patients found significant mortality reduction from HEMS.[11] Another study from southern Texas, which compared survival rates before and after the cessation of HEMS programs, reached the opposite conclusion.[12] A five-year study of blunt-trauma patients in an urban setting with a sophisticated prehospital care system transported either by HEMs or ground ambulance found no survival advantage for patients transported by HEMS.[13]

Similar conclusions have been reached about HEMS by investigators in Great Britain, Australia, Norway, Italy, and Hong Kong, where HEMS is felt also to benefit a small fraction of those transported.[14-17]

The use of HEMS for children has also been evaluated. A Los Angeles study of 189 pediatric trauma patients transported by HEMS found that 57 (33%) were discharged from the ED.[19] These authors concluded that HEMS for pediatric patients was associated with a high rate of over-triage, with no additional prehosptial interventions, when compared with ground transport. A Washington, D.C., study of nearly 4,000 pediatric trauma patients transported by HEMS found that nearly 85% were over-triaged and did not require HEMS.[19] Another study from New Jersey evaluated the utilization of HEMS for pediatric trauma patients. The authors found that pediatric patients transported by HEMS were equally severely injured as were those transported by ground, in contrast to adult patients, for whom the HEMS was reserved for the most severely injured patients. They speculated that, because trauma triage schemes were designed primarily for adults, ground personnel are more selective about which adult patients are flown to a trauma center and less selective for children. They recommended the development of pediatric trauma triage protocols.[20]

It has been estimated that approximately 28% of the US population has access to Level I or Level II trauma center care within an hour only by helicopter. The discontinuation of HEMS was found to have a detrimental impact on mortality in one system in interfacility transport to a tertiary trauma center.[21]

Noise levels in most helicopters while in flight prevent accurate physical examination and auscultation and are a limiting factor for managing patients. Airway interventions are often difficult or impossible in flight, and, if the patient deteriorates en route, management may be extremely difficult.

Medical helicopters are estimated to cost between $1,500,000 and $5,700,000, depending on their configuration and equipment.[2,23] The annual cost for operating a helicopter is estimated to be approximately $1 million.[13] It is estimated that each helicopter requires five hours of maintenance for each hour it flies.[22] The HEMS system at the University of Michigan had operational costs of $6 million, but generated $62 million in inpatient revenues and 28% of ICU days. HEMS patients were also twice as likely to have commercial insurance as were other patients.[2] Some authors have speculated that the proliferation of HEMS is a direct result of successful negotiation for favorable rates of reimbursement. In 2004, the number of flights paid for by Medicare was 58% higher than three years earlier. Spending for HEMS by Medicare more than doubled to $103 million over the same period. In 2002, Medicare increased the rates for HEMS, with prices of from $5,000 to $10,000 per flight, or five to ten times the rate for ground transport. In one study of adult cost per life-year saved, HEMS was calculated to be about $2,500. This compares to $18,000 for neonatal ICU stays for birth weights 500-9999 g; $19,000 median for 310 medical interventions; $23,000 for three-vessel coronary artery bypass for severe angina; $32,678 for thrombolytic therapy for acute MI; and $41,000 for AZT prophylaxis after needle stick.[24] In a 2002 Finish study that dealt with HEMS transport for both trauma and non-trauma diagnoses however, the cost per life-year saved was $30,000.[14]

In addition to the debates about the efficacy and expense of HEMS is the issue of safety. As HEMS usage has increased, so has the number of helicopter crashes. There was a steady and marked increase in the number of HEMS accidents in the United States from 1993 to 2002. Over this period, there were 84 crashes involving 260 people (passengers, patients, crew, and pilots). Of these, there were 72 deaths and 64 injuries. Fifty-two percent of the accidents occurred during the last three years of the study.[25] HEMS aircraft have killed 28 people in seven separate accidents in 2008, which has been the deadliest year for EMS helicopter crashes to date. Since 1987, more than 200 EMS helicopters have crashed, killing 202 people, (Figure 1). This year's fatalities are twice as many as any past year, according to the Comprehensive Medical Aviation Safety Database, a product of Humanitarian Research Services.

In 2001, there were 12 fatalities per million flight hours for all helicopters, but there were 19 per million for HEMS. Between 1995 and 2001, the rate of occupational deaths per 100,000 was five for all workers, 26 for farmers, 27 for miners, but 74 for HEMS crew. At this rate, it is estimated that a HEMS pilot or

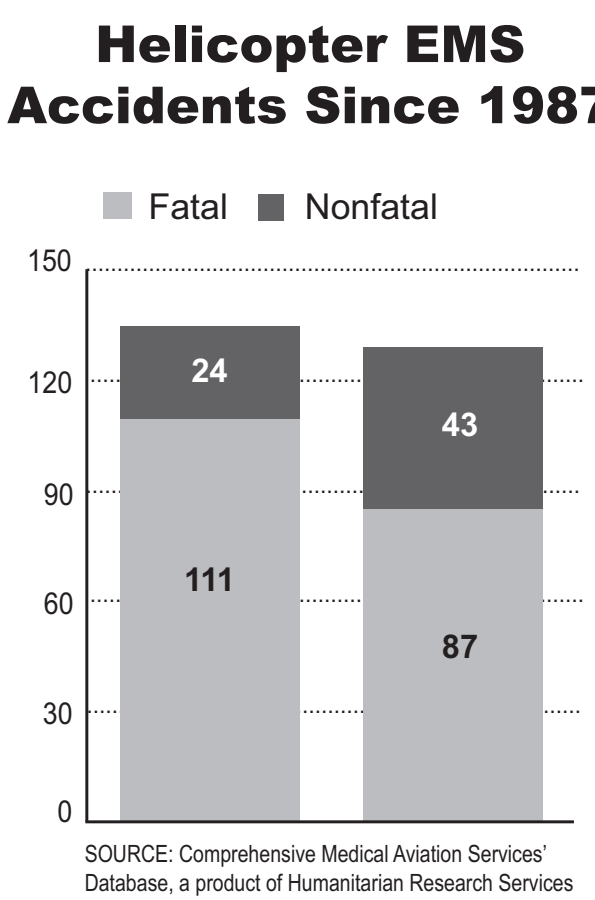


Figure 1: Helicopter EMS crashes since 1987

crew flying 20 hours per week for 20 years would have a 40% chance of a fatal crash. Thirty-nine percent of all HEMS crashes result in one or more fatalities. HEMS crashes are associated with post-crash fire and often occur in darkness or bad weather. Darkness more than triples the risk of fatalities, and bad weather increases the risk eight-fold.[26] According to National Highway Transportation (NHTSA) data, causes of HEMS crashes in decreasing order are pilot error, mechanical failure, and undetermined causes.[25] A 2001, HEMS survey found that many pilots felt that they were under unreasonable pressure from management, dispatchers, and flight crews to speed response or lift-off times, to fly when fatigued or ill, and to launch or continue flights in marginal conditions.[27] Medical specialists in sending and receiving hospitals also may feel increased pressure from hospital management to use helicopter services when ground transport would have sufficed.

The Injury Severity Score (ISS), Revised Trauma Score, (RTS) and the Trauma Injury Severity Score

(TRISS) have all been investigated as predictors of the need for HEMS in trauma. It has been observed that patients whose injury severity is in the mid-range of the bell-shaped distribution of trauma severity (i.e., those with ISS between 15 and 45 to 60) are the ones most likely to benefit from HEMS. In studies from North Carolina and Pennsylvania, those patients with an ISS between 15 and 40 had the clearest benefit of HEMS.[28,29] In a Boston area study of patients with moderate-to-severe trauma, HEMS was felt to confer a 24% mortality reduction in a similar group of patients who were transported by ground EMS. The mechanism of injury alone has been found to be a poor indicator of who benefits from HEMS.[30]

HEMS operational safety could be improved by using stricter weather guidelines, medical-necessity algorithms, and standardized fly/no-fly protocols for pilots. Also suggested are instrument flight rules, night-vision devices, dual pilots, and enhanced minimum pilot qualifications. Regional triage guidelines for HEMS should be established and followed.[14,26,30, 32-4] Data-driven and team-based utilization review of the appropriateness of the transport should also take place. This review must be nearly concurrent with the flight, and it must be applied to all flights. Overtriage, (the use of HEMS to transport patients who are not critically ill or injured) and undertriage (the failure to use HEMS to transport patients who are critical) are two measures used to judge the appropriateness of HEMS. Trauma centers have a built-in accommodation for an overtriage rate of up to 50% in order to have an acceptable undertriage rate (often quoted to be up to 10%).[14,30]

Current medical literature and EMS experts have suggested that HEMS resources might be better allocated by following the Australian and German models of state-run, rather than hospital-owned and based, services. In the US, HEMS operation as part of an EMS or governmental entity (e.g., Maryland State Police) might also improve appropriate usage. As stated above, a HEMS usage criterion, based on physiological parameters rather than on mechanism of injury, has also been recommended. Also, by concentrating on rural responses rather than inter-hospital transfers and urban responses, resources might be better allocated.[2,30]

One HEMS authority, Dr. Thom Mayer of Inova Fairfax Hospital in Virginia, perceptively observes that, "it's not how long it takes the patient to reach the regional critical care center, but rather how long it takes the resources of the regional critical care center to reach the patient. In this respect, the critical care flight crew is an extension of the regional center and may be a key determinant of outcome." He has stated that in his EMS system, HEMS is appropriate if the patient at the scene would have warranted a trauma code if he or she were in his hospital's emergency department (e.g., airway emergency, BP <90 with signs of shock, GCS < 8, traumatic paralysis, penetrating head, neck, or torso injury, major crush or amputation, compartment syndrome, > 20% BSA burn, and extremes of age), unless the patient is less than five minutes from the trauma center by ground ambulance.[30]

It was estimated in one study that, in a mixed rural and urban EMS system, one may convert air and ground miles estimates by using the relationship: lights and sirens ground miles = 1.3 X air miles. The authors suggested the use of this conversion coefficient in designing reasonable helicopter utilization policies.[31]

The considerable debate on the appropriate use of HEMS will continue, and it will be intensified by spiraling health care costs, increasing medico-legal scrutiny, the recent uptick in crashes, and by health care market forces, among many other competing pressures.[32-35]

Ambulance Light and Siren Use

The use of lights and siren (L&S) during ambulance transport of critically ill and injured patients (a.k.a. "Code 3" transport) is commonly employed by EMS systems. Prominent among reasons cited for using L&S are critical patient status and critical system status, in which there is pressure to return the ambulance rapidly to be available for other calls. It is clear that the indiscriminate use of L&S is a significant contributing factor in ambulance crashes. It has been estimated by a variety of sources that Code 3 status is warranted in only approximately 5% of ambulance runs. Empirical data confirm that the use of L&S far exceeds this estimate.

A 1994 study evaluated the use of a protocol that limited Code 3 transport in Pennsylvania. Before implementation of the protocol, L&S were employed in 58 % of ambulance runs.[36] Use of the protocol reduced Code 3 transport to 8% of ambulance runs. Of the remaining 92% of patients transported without L&S, no patient was judged to have sustained an adverse outcome related to transport mode. A New York study of L&S transport found that their use only reduced response times by an average of 106 seconds.[37] These authors concluded that, "Although statistically significant, this time savings is likely to be clinically relevant in only a few cases." A similar study conducted in Minnesota over a nine-month period found that L&S use decreased response time an average of 3.02 minutes compared to non-L&S use.[38] A North Carolina study compared transport times of EMS with L&S and without, when the trip length was less than eight miles. The authors found that L&S saved an average of 43.5 seconds per trip, and they concluded that, "Although the mean difference is statistically significant, it is not clinically significant, except in rare circumstances."[39]

In a recent Pennsylvania study of 245 consecutive patients who arrived by Code 3 transport, only 14% received an ED intervention within 15 minutes of arrival, and only 54% were ultimately admitted to the hospital.[40] In Cincinnati, L&S were employed in nearly two-thirds of over 500 ambulance runs to a pediatric medical center. The use of L&S was considered to be inappropriate in 39% of the runs, and its use was more common in basic ambulance units than in paramedic units. The authors concluded that L&S transport of pediatric patients in their system was often inappropriate and that protocols should be established to limit L&S use.[41]

A retrospective study of data from the Rapid Early Action for Coronary Treatment (REACT) trial compared the mode of transport of chest pain patients in 20 US cities. Patients transported by private transportation arrived more quickly than those who arrived by ambulance (35 minutes vs. 39 minutes). The authors noted that, although activating 9-1-1 is the most rapid way to achieve definitive medical care, only 50%-60% of patients with chest pain choose to initiate care via EMS, principally because of their perception that private transportation is quicker. "Door-to-needle" time was faster in the EMS patients, however (32 vs. 49 minutes).[42]

The unrestricted use of L&S is not only medically inappropriate, but it is also dangerous. It has been estimated that 12,000 EMS crashes result in 120 deaths in the United States and Canada annually. Most of these crashes are associated with the use of L&S and involve more frequent and more severe injuries than are sustained in crashes in non-Code 3 transport.[41-44] As a result of these increased injuries, liability claims are more than 20 times more likely to result from EMS vehicle crashes than are claims involving the EMS patient care. In 2004, 170 fatalities in the US occurred as a result of emergency vehicle crashes, according to NHTSA.

During the years 1991-2000, the MMWR reported 300 fatal crashes involving occupied ambulances, with 82 deaths of ambulance occupants and 275 occupants of other vehicles and pedestrians.[44] A total of 816 ambulance occupants were involved in these 300 crashes. Twenty-seven of the occupant fatalities were on-duty EMS workers, representing 3% of all ambulance occupants and 33% of occupant fatalities. Most of the 27 EMS worker fatalities occurred in the front of the vehicles. Riding or driving unrestrained was cited as a major contributor to death and injury. Less than half of the EMS workers in the rear compartments use restraints, often citing unsatisfactory access to the patient for IV insertion, CPR, and airway management. Additionally, unrestrained patients in ambulance crashes have the potential to become airborne and to endanger other rear compartment occupants. Three times as many bystanders (either pedestrians or occupants of other vehicles) were killed as were EMS personnel.[42,44]

EMS workers in the United States have a fatality rate of 12.7 per 100,000 workers, more than twice the national average, and most of these fatalities are due to vehicle crashes. By comparison, the fatality rate for police is 14.2, and it is 16.5 for firefighters.[44, 45]

Also frequently cited in ambulance crash literature is the "wake effect" – the tendency for the racing Code 3 ambulance to precipitate crashes of other vehicles in its wake. A study from Salt Lake City substantiated the existence and magnitude of wake-effect collisions. Sixty ambulance crashes and 255 wake-effect collisions were reported. The study suggested that wake-effect collisions are real and that these probably occur with a greater frequency than do ambulance crashes.[46]

Restriction of the use of L&S to a prearranged set of indications is likely to minimize ambulance personnel injury. The National Association of EMS Physicians (NAEMSP) and other organizations have policies regarding the prudent use of L&S. It is clear that personnel in many EMS and fire systems feel inadequately trained in vehicle operation safety, especially when compared to their counterparts in law enforcement.

It is also evident that EMS personnel knowledge is poor regarding basic traffic safety laws pertaining to emergency vehicle operation.[47] In a sample of 293 EMTs at East Carolina University, the median number of correct responses to five knowledge questions about ambulance operation was one. The median number of correct responses to the five knowledge questions was one (range zero to four). Thirty-three percent of the EMTs knew that other vehicles are required by law to yield while either approaching or being overtaken by an ambulance with warning lights and sirens; 2% knew that due regard for safety is the only requirement of an ambulance approaching a red light at an intersection; 14% knew that the minimum following distance behind an ambulance is one city block; and 28% knew that there is no speed limit on ambulances with warning lights and sirens. Respondents were more likely to score above the median if they had taken one or more emergency driver's education courses or had nine years or more of EMS experience.[48]

In a review from Virginia, while 75% of ambulance runs were conducted with L&S, a disproportionate number (91%) of collisions occurred during L&S operation. The responding ambulance driver had a history of multiple EMS crashes in 71% of the collisions.[49,50]

All operators and front-seat passengers of ambulances must use seat belts. Any patient on a stretcher must be secured while the vehicle is in motion, and all EMS personnel in the patient compartment must use seat belts when not attending to the patient. It is unrealistic to expect the public to use seat belts if healthcare workers fail to use them.

Prehospital Analgesia

In the words of Albert Schweitzer, "We must all die. But that I can save a person from days of torture, that is what I feel is my great and ever-new privilege. Pain is a more terrible lord of mankind than even death itself."

While the goal of medicine is largely to decrease pain and suffering, pain management in EMS continues to be woefully inadequate. This is despite the observation that up to 70% of our patients experience pain as part of their presenting problem.[49] Among the organizations that have position papers on the use of analgesia in EMS are the National Association of EMS Physicians, the American College of Emergency Physicians, the American Academy of Pediatrics, the American Medical Association, the American College of Obstetrics and Gynecology, and the American College of Surgeons.

One study showed that up to 20% of EMS patients have moderate-to-severe pain.[51] Other studies have revealed that medical practitioners in general and EMS workers in particular are poor at recognizing and managing pain. In one EMS study of 1,073 patients with suspected extremity fractures, only 1.8% were administered analgesics and 17% and 25% received ice packs and air splints, respectively.[52] In another study of 124 patients with an emergency department diagnosis of hip or lower extremity fractures, only 18.3% were administrated field analgesics. In Australia, of 128 patients with a prehospital diagnosis of femoral neck fractures, only 51% received analgesics by EMS.[53,54] In addition to the infrequent administration of analgesia by prehospital personnel, the patients who receive their first analgesia after arrival at the emergency department wait much longer to receive them. In one study, this time was 28 minutes vs. 146 minutes on average.[55] In a second study, the time was 23 minutes and 113 minutes respectively.[56] There are several barriers to adequate analgesia in EMS. The first is that many states still require physician contact before the administration of narcotics. Next, few EMS textbooks devote significant attention to analgesia, and EMS education is often inadequate in this field. Also, there are many EMS systems that have no written protocol for analgesic administration. In addition, there is often reluctance by EMS personnel to administer analgesia for fear of conflict with the emergency physician. Lack of education and research and of agent availability are also cited.[57] Prejudices about EMS analgesia administration may include a belief that its use may mask important physical exam findings and that it may lead to addiction. EMS care providers overestimate their abilities to accurately assess a patient's pain by observation alone. There is also an unfounded concern that analgesic administration might make later informed consent impossible. Also cited is a fear of regulatory oversight and misunderstanding about the likelihood of adverse events.[56] Ethnicity of the patient has also been shown to affect pain management. A UCLA study showed that Latino patients with isolated long bone fractures were half as likely to receive pain medication as were their non-Latino white counterparts.[57] A New Orleans study showed the same finding for African-Americans.[58] Children and adolescents have been shown to have less documentation of pain assessment by EMS personnel and to be less likely to receive analgesia.[59,60] Women have been shown to be less likely than men to receive prehospital analgesia for isolated extremity injuries.[61] Decreasing levels of income are also associated with decreased rates of analgesia administration.[56,61,62]

Among the most accurate means of pain assessment by EMS providers is self-reporting by the patient. In addition to this, visual analog scores, numeric pain scales, and pediatric FACES pain scales are useful in measuring pain degree and of its responsiveness to analgesics. Multiple studies have demonstrated that narcotic analgesics actually make subsequent abdominal examinations more accurate. Further, it has never been shown that analgesics given judiciously for legitimate pain interfere with informed consent or that they lead to drug addiction.[63] Several reports have demonstrated the safety of EMS narcotic administration.[64,65] In one such study of 84 cases using small intravenous doses of morphine (2-4 mg), there was only one case of respiratory depression. In another study of 131 HEMS patients there were no complications from intravenous fentanyl administration. Of another cohort of 2,129 patients who received intravenous fentanyl by EMS, 12 patients (0.6%) had a transient vital sign abnormality and none required any intervention.[65] Thirty-seven states allow standing orders for narcotic analgesic administration, and 16 states endorse standing orders for fentanyl for pain management for extremity fractures and burns.

The ideal EMS analgesic has a short onset of action and time to peak effect and a short duration. It causes minimal hypotension, respiratory suppression, and nausea. It is easy to administer, is inexpensive, reversible, and it has multiple routes of administration. Fentanyl citrate (Sublimaze) is one such medication.[65] It has intravenous, intramuscular, intranasal, transmucosal, and transdermal routes, and it may be used in adults and children.[65]

Several other medications have been studied in EMS and have been found to be efficacious: nitrous oxide (Entonox) and methoxyflurane inhalers, morphine sulfate, tramadol (Ultram), butorphenol (Stadol), ketamine (Ketalar), and alfentanyl (Alfenta).[66-76] Non-pharmacological interventions have also been studied and have been found to be effective. Among these are guided imagery, biofeedback, breathing exercises, emotional support, splinting and positioning, elevation, and ice or heat. In one EMS study, patients who received acupressure were found to have less pain, less anxiety, a slower heart rate, and greater satisfaction than did patients in a control group.[77]

Among measures that have improved prehospital pain control is the use of objective pain instruments in the assessment of the presence and degree of pain.[78,79] By the agreement of EMS physicians and field personnel, administrators, and receiving hospital personnel, a comprehensive prehospital pain plan would liberalize protocols and move most real-time pain management decisions from on-line medical control to written protocols. Such a protocol was found to reduce the time to morphine administration by 2.3 minutes in one study of isolated extremity fractures.[80]

In summary, prehospital pain management can be performed safely when appropriate drug choices, protocols, education, documentation, and quality management tools are integrated. Only by emphasis on pain education, research, protocols, and monitoring will the assessment and management of pain in the prehospital setting improve. This is both humane as well as being good medicine.

EMS Airway Management

Among the most hotly debated issues in EMS currently is optimal airway management. With the introduction of paramedics in the 1970s, increasingly sophisticated airway care was available in the field. In a series of reports in the literature from the 1970s and 1980s, it seemed that prehospital endotracheal intubation (ETI) was feasible and effective, and it has largely come to be considered the standard of prehospital airway care. In theory, ETI achieves tight regulation of oxygenation and ventilation, protects against aspiration, allows suctioning, and provides an alternate route for drug administration.

In 2001, Katz and colleagues took a fresh look at ETI in his EMS system.[81] For many, the results were shocking. Fully one-quarter of the endotracheal tubes were misplaced, (i.e., tip of ET tube above the cords in the hypopharynx, or in the esophagus). In another report in 2003 from Maine by Jemmett et al., prehospital endotracheal tubes were misplaced at a rate of 12-15%[82] In a retrospective review of over 4,000 with severely head injured patients in Pennsylvania, 44% of patients were intubated in the prehospital setting, and the rest were intubated after arrival in the emergency department. The adjusted odds ratio for death for the patients receiving prehospital intubation was 3.99. Prehospital intubation was also associated with worse neurological and functional outcomes: 18.2% vs. 15.5%, respectively.[83]

In a study of 8,786 adult trauma patients, prehospital ETI and positive pressure ventilation in severely injured adults (GCS of 8 or lower, and an ISS of 16 or higher) was associated with an increased risk of early hypotension and an increased mortality.[84] In another series of 852 patients with severe head injury (GCS < 8) who were admitted to one of 13 trauma centers from 1995 to 1997, the relative risk of mortality was 1.74 in intubated patients and 1.53 in patients undergoing unsuccessful intubation attempts. A "Best Evidence Topic Report" from the Emergency Medicine Journal examined whether prehospital ETI was superior to bag-valve-mask (BVM) ventilation in 17,676 patients in eight relevant papers.[85] The authors found that patients undergoing ETI had longer prehospital times as well as higher mortality when compared to the BVM group. A 2003 study from the R. Adams Cowley Shock Trauma Center in Baltimore prospectively studied 191 severely head-injured adult patients who survived at least 48 hours after admission. In comparison to the 59% of patients receiving BVM in the field, those 41% who were intubated in the field had a longer mean duration of mechanical ventilation, longer hospital stays, an increased rate of pneumonia, and a higher mortality (23% vs. 12.4%). In a 2005 study, 13,625 moderate-to severely-brain injured patients were studied.[86] Prehospital ETI was performed in approximately one-fifth of the patients. Intubated patients had a mortality rate of 55% compared with 15% in those without prehospital ETI.

Gausche and coworkers at Harbor-UCLA Medical Center in Los Angeles published a three-year study of 830 pediatric patients comparing survival and neurological outcomes of prehospital BVM vs. ETI.[87] More than 2,500 paramedics in Los Angeles and Orange Counties received intensive pediatric airway training prior to the study. The study found no significant difference in survival or in achieving a good neurological outcome among children receiving either procedure. This was the first controlled study comparing the widely used BVM and ETI treatments in either adults or children and is the longest and largest controlled trial of treatments for children in a prehospital setting to date. BVM was found to be as effective as ETI in an urban EMS system. The study also demonstrated increased scene times and overall times when ETI was used. ETI was associated with a significant rate (8%) of fatal complications. Children are especially susceptible to tube dislodgements due to their short tracheal lengths. Therefore, once an endotracheal tube was placed, there was a significant risk of dislodgement, which occurred in 14% of cases. According to Dr. Gausche, "It is clear to me that the best way to manage a child's airway in the field who require ventilatory support is via BVM ventilation."

In the San Diego Rapid Sequence Intubation (RSI) trial, Dunford et al. found that oxygen desaturation (SaO2 <90%) occurred in over half of cases, bradycardia (heart rate < 50 beats per minute) occurred in 19%.[88] Despite this, paramedics described the intubations as "easy" in 84% of the cases in which desaturation occurred. The RSI group had lower rates of "good outcomes," longer scene times, and more frequent inadvertent hyperventilation, when compared to a control group. Fifty percent of the RSI group experienced transient hypoxia. It will be noted that, in traumatic brain injury, the combination of hypoxia and hypocapnea is a recipe for secondary brain injury. The PACE II trial studied 1,953 prehospital intubations in over 40 EMS agencies in Pennsylvania. In these intubations, 22.7% (of 1 in 4.5) patients were exposed to at least one of three errors: 1) tube misplacement or dislodgement, 2) multiple attempts defined as four or more laryngoscopies, and 3) intubation failure. There was significant variability in intubation success rates between agencies, with some experiencing error rates as high as 30-40%.[89]

Another study from San Diego evaluated the relationship between hypoxia and increased mortality in 13,625 patients with moderate to severe traumatic brain injury. The mortality rate was 55% for patients undergoing prehospital ETI compared with 15% for those without invasive airway management. In two other studies, targeted ventilation rates in traumatic brain injury patients were associated with lower mortality when compared to hyper- or hypo-ventilation.[90]

In a 2005 Texas study, prehospital ETI and positive pressure ventilation in severely injured adults was associated with an increase in hypotension upon arrival in the ED (54% vs. 33%) and decreased survival (24% vs. 45%).[90]

An observational prospective study at the Indiana University School of Medicine found that, of 208 consecutively enrolled patients who were intubated in the field (77% medical and 23% trauma), 5% of orotracheal, and 11% of nasotracheal ETIs were misplaced.[91] Stringent paramedic training requirements and close medical direction were cited by the authors as possible reasons for these lower rates of misplaced ETIs when compared with other current studies.

A significant complication of ETI is the aspiration of gastric contents. One new and ingenious method of detecting gastric aspiration after ETI was by described by Ufberg et al. at Temple University.[93] They tested sputum specimens obtained after ETI for the presence of pepsin, a marker of gastric contents. From pepsin, they were able to determine the rate of aspiration in the prehospital setting. Their conclusion was that prehospital ETI was associated with aspiration with an odds ratio of 3.5 when compared with ED intubation. In a second study, Ufberg et al. went on to show that aspiration syndrome was present in more than half of pepsin assay-positive patients vs. 21% of assay-negative patients and that death occurred in patients with aspiration syndrome in 44% vs. 12% in patients without the syndrome. Thus aspiration syndrome after emergent intubation was strongly associated with death during hospitalization.[94]

The gold standard in assuring adequate ventilation in emergency departments and operating rooms is waveform CO_2 capnography.[95] This monitoring technique is not generally available in the prehospital setting, where tube placement is usually confirmed by a combination of other techniques: esophageal detector device, direct laryngoscopic visualization, and colorimetric CO_2 detector.[96,97] In one report, the use of end-tidal CO_2 monitoring decreased the rate of endotracheal tube misplacement from 23.3% to 0%. Eventually, waveform CO_2 capnography is likely to become the standard of care in the field.[98]

Another promising new device in prehospital airway management is the airway impedance device (ITD, Res-Q-Pod, Advanced Circulatory Systems, Inc. Eden Prairie, MN. This device is recommended as a Class IIa device in the 2005 American Heart Association CPR Guidelines, and thus is more highly recommended than any other device or drug used by emergency personnel for increasing circulation during CPR and for improving resuscitation rates.[108] The ITD is introduced between the endotracheal tube and bag-valve and is intended to prevent over-bagging of intubated patients, thus addressing the hazards of increased intrathoracic pressure and impeded venous return. In several reports this device has been found to increase systolic pressures safely and significantly in patients in cardiac arrest compared with sham controls, thus increasing blood flow to the heart and brain during assisted ventilation. It has been shown to be effective with standard CPR and with other methods of CPR (i.e., active compression decompression - ACD).[109] It is hoped that its use will increase the rates of survival and normal neurological function after cardiac arrest.

In addition to the problems created by over-bagging of intubated patients are the problems caused by over-inflation of the endotracheal tube (ETT) cuff. Such over-inflation can lead to severe complications, such as tracheal necrosis, laryngeal nerve palsy, and tracheoesophageal fistulas. Under-inflation can lead to air leaks, inadequate ventilation, and aspiration. In one study, every one of 53 experienced paramedics inflated the ETT cuff over the safe pressure limit of 25 cm H_2O. In 66% of the cases, the ETT cuff pressure was over 120 cm H_2O, and 87% of paramedics could not detect an over-inflated ETT cuff by palpation. The authors recommended use of commercial ETT cuff inflation devices to achieve optimal pressures and the checking of cuff pressure with a manometer.[110]

A potential solution to the problem of prehospital intubation failures and complications is to use alternative airways such as the Combitube, Laryngeal Mask Airway, and the King LT airway (King Systems Corporation). Regardless of the solutions chosen to avoid airway complications in EMS, it is clear that the airway is the paramount consideration in resuscitation and rescue. Misplaced airways are a problem. Undetected misplaced airways are disasters. Any steps that can assure a secure and functional rescue airway in EMS must be adopted immediately.[111-115]

Cardiopulmonary Resuscitation and Advanced Cardiac Life Support

Sudden cardiac death is a major public health problem affecting 400,000 patients annually in the United States, with the majority of these occurring in the out-of-hospital setting.[116] Mortality rates are high and reach almost 100% when prehospital care has failed to restore spontaneous circulation. Overall survival remains at approximately 5% in most communities. Of the survivors, only about two-thirds have good neurological function. Advanced cardiac life support (ACLS) is the "fourth link" in the American Heart Association's "chain of survival": early EMS care, early CPR, early defibrillation, and ACLS. But among the more disturbing recent revelations about the effectiveness of ACLS was a 2004 report by Stiell and colleagues that appeared in the New England Journal of Medicine.[117] The authors, members of the Ontario Prehospital Advanced Life Support (OPALS) trial, evaluated the contribution of paramedic-provided ACLS care to survival. Patients treated in an initial rapid defibrillation cohort were compared to a later group who also had prehospital ACLS care. During the second (ACLS) phase, although there was a significant increase in the rate of return of spontaneous circulation and of survival to hospital admission, there was no increase in survival to discharge. The authors concluded that resources should be concentrated on increasing bystander CPR and early defibrillation rather than on prehospital ACLS (e.g., intubation, medications). For prehospital respiratory (as opposed to cardiac) distress, the OPALS investigators found that there was a decrease in mortality with the introduction of an advanced life support program, even though ACLS interventions were rarely used.[118] Other symptomatic treatments, such as nebulized albuterol and sublingual nitroglycerine, were added to an existing basic life support system simultaneously with the ACLS measures of ETI and intravenous medications. The contribution of the ALS measures to the overall benefit to respiratory distress patients could not be determined in this study, but the ALS group mortality was 14.3% vs. 12.4% in the pre-ALS group.

The effect of advanced life support on survival in children sustaining out-of-hospital cardiac arrest (OOHCA) has also been studied. In a 2002 retrospective chart review from Children's Hospital of Pittsburgh, survival rates of children in cardiac arrest who received basic life support (i.e., BVM ventilation) were compared with those receiving advanced life support (i.e., intubation, defibrillation, epinephrine, bicarbonate, atropine). There were no significant differences between the two groups in survival to hospital discharge.[119]

In both the OPALS and other studies, the most important predictors of survival to discharge of OOHCA were arrest witnessed by a bystander, early CPR, and early defibrillation. Valenzuela and colleagues showed that time to defibrillation was strongly correlated with survival.[120] Survival with defibrillation at nine minutes was 4.6%, eight minutes 5.9%, seven minutes 7.5%, six minutes 9.5%, and five minutes 12.0%. In the OPALS trial, defibrillation before eight minutes correlated with an odds ratio of survival of 3.4.

One fundamental assumption about CPR that may be erroneous is that the addition of ventilation to chest compression is necessary. In a well designed 2007 study that was published in Lancet, Nagao et al. found that CPR done with chest compressions only was just as effective as that performed with ventilation.[120] In this prospective observational study of 4,068 adult survivors of OOHCA, 71% had no bystander CPR, and these patients had a 2% positive neurological outcome. Of the remaining 29% of patients who received bystander CPR, 11% had compression-only resuscitation and 18% had conventional CPR with ventilation and compression. There was a favorable neurological outcome at 30 days of 5% in both of these groups, with no differences in survival. Given the possibility that bystanders in an OOHCA might be reluctant to perform mouth-to-mouth ventilations, this research supports the efficacy of omitting artificial respiration.

Wik and colleagues investigated whether defibrillation should always be attempted first or whether it should be preceded by a period of CPR. They found that when defibrillation was delayed more than five minutes, there was an improvement in ROSC in patients who received chest compression for three minutes prior to defibrillation (58% vs. 38%).[121] In another study, 90 seconds of CPR prior to defibrillation resulted in higher survival if EMS response time exceeded four minutes.[121-123]

Wik et al. also evaluated the quality of CPR during OOHCA in 176 adults in England, Sweden, and Norway, and found that a large proportion of CPR was performed poorly.[121-123] Chest compressions were not performed 48% of the elapsed time that there was no spontaneous circulation, (38% if accounting for time to evaluate the EKG and for defibrillations). Further, the mean compression depth was 34 mm, compared with the recommended depth of 38 to 51 mm. Similar results were obtained in a University of Chicago study, which found that chest compressions were performed too slowly and too shallowly, that ventilation rate was too fast, and that too long a period of no compression took place for CPR to be effective in many cases. Thus, the poor quality of CPR itself may have much to do with the persistently dismal (5-10%) survival rate after CPR that is often quoted.[127]

The relationship of survival and EMS response times has been evaluated. Papers by Pons et al. and Blackwell et al. have questioned the generally-accepted national benchmark of eight minutes that is used in most urban areas.[128,129] It is known from the OPALS data that survival after cardiac arrest declines dramatically after five minutes,[122] yet the National Fire Protection Association has set its target for communities to "provide for the arrival of an ALS company within an eight-minute response time in 90% of incidents." But in practice the response time target is nine minutes, not eight, since the benchmark actually strives for nine minutes zero seconds with 90% reliability. This response time is obviously not likely to improve survival in OOHCA (see discussion of AED deployment below).[130]

It is widely accepted that, for each minute a patient remains in VF and defibrillation is not provided, the chances for survival

drop by almost 10%. Further, after ten minutes, the chances for resuscitation are near zero. Considering the pivotal role of defibrillation in survival of OOHCA, public access defibrillation has been intensively investigated. The PAD trial was a multicenter study sponsored by the National Institutes of Health, in which intensive public education was combined with AED installation in "high risk" settings. These included a total of 1,250 places where there were usually more than 250 people over 50 years of age for most of each day, and places where OOHCA had occurred within the past two years. The primary endpoint was survival to discharge. Based on preliminary data, approximately 10-15 lives were saved, at a cost of well over $100 million.[129-130]

Several studies have identified locations for automated external defibrillators (AEDs) that have been associated with early successful defibrillation.[131-135] These include gaming casinos, airports, nursing homes, and dialysis clinics, among others. Of note, the particular locations where AEDs appear to be cost-effective vary from one country and one community to another. Since 80% of OOHCAs are estimated to take place in the home, the value of a more generalized availability of AEDs in the public domain is currently being studied.[131] The cost-effectiveness and feasibility of AEDs in the home remains unproven; one study showed that survival in residential AED use was only 3.3%.[131] A multicenter North American study examined the effect of AEDs on the likelihood of survival to hospital discharge in OOHCA.[132] Of nine hundred ninety-three units, 85% were placed in a public place, primarily in recreational facilities and shopping malls. The remaining AEDs were placed in patients' homes. The study compared outcome for a lay CPR-only group to that for CPR-plus-AED. There was a 14% survival in the CPR-only group and a 23% survival in the CPR-plus-AED group. Of the survivors, almost all arrests occurred in an area served by the public, rather than the residential, AEDs. A number of communities have equipped police as well as other first-responders with AEDs.[133,134] In Pittsburgh, 183 EMS resuscitations were compared to 118 police-applied AEDs.[135] Mean time to defibrillation decreased from 11.8 minutes in the EMS group to 8.7 minutes in the police group. The earlier shock in the police group was an independent predictor of survival to hospital discharge. Another study from the same authors reviewed ten years of police AED use. Overall, 77% of officers had used an AED, and 45% had witnessed return of spontaneous circulation prior to EMS arrival. Most (65%) did not feel that AED use interfered with other police duties. But all communities are not the same. In a study conducted in suburban and rural Indiana, a police AED program was compared to a standard EMS response. Mean time to arrival by equipping the police with AEDs on scene decreased by 1.6 minutes. Time to first shock decreased by 4.8 minutes. Despite the shorter response and defibrillation times in the police group, survival to hospital discharge was not improved in this study.[136] The author concluded that the lack of improvement in survival was related in part at least to a very limited response to out-of-the hospital cardiac arrest by police officers. Despite having almost half the defibrillator capability in their counties, police responded for traditional EMS in only 6.7 percent of cases. When asked, almost half of the police admitted that they were uncomfortable in the role of treating people in cardiac arrest. They also told investigators that other responsibilities and long travel distances decreased the likelihood that they would respond.

In a report from one suburban community's experience with police AEDs over seven years, survival to discharge for the police group was 9.9% vs. 11.9% in the ALS group, and time to defibrillation was 6.6 minutes vs. 8.4 minutes, respectively. In this study, cost per life-year saved was estimated to range from $1,582 to $16,060, which would be more cost effective than many other standard medical therapies.[136,137]

Authors from Scotland investigated the clinical effectiveness, public health impact, and cost-effectiveness of PAD.[138] Citing a recommendation by the American Heart Association to place an AED in locations where there is an expected rate of one cardiac arrest per defibrillator per five years, these authors estimated that AEDs would only address 1-2% of OOHCAs and would have a minimal impact on population survival and may represent poorer value for money than other interventions.

In another estimate of the cost-effectiveness of AEDs in high-incidence environments (airports, airplanes, casinos), the cost would be "less than the typically acceptable $50,000 per quality-adjusted life- year." The authors, epidemiologists from the University of Washington, concluded that AEDs appear to be cost-effective in locations with high incidences of cardiac arrest.[128-138]

The above data, regarding the crucial role of early defibrillation in survival and the lack of apparent benefit of ALS measures, raise important questions about the best way to allocate EMS resources. Might EMT-Ds (defibrillation) and other first responders (e.g., police, firefighters) prove more cost-effective than paramedic units in improving outcomes of OOHCA? Individual communities and EMS systems will have to weight the evidence and apply it to their own circumstances, but there is ample reason to question our current practices.

Summary

Much of what is currently believed about prehospital care is based on custom and tradition rather than on sound scientific evidence. As our healthcare dollar is stretched to a breaking point, it becomes increasingly crucial that we evaluate the costs and benefits of EMS care in a dispassionate and critical way.

Recent clinical studies suggest that helicopters and ambulance lights and sirens are overused. Further, cardiopulmonary resuscitation is performed poorly and rescue breathing may not be required. Defibrillation is performed too late to benefit patients in many cases. AEDs used by first responders and by the public may be more effective than later defibrillation by paramedics. Pain is managed poorly, if at all, in the prehospital setting.

Emergency physicians and EMS directors are in a unique position at the interface of prehospital and hospital care, and they

are the stewards of a precious and finite set of resources upon which the public safety depends. It is sincerely hoped that the bright light of scientific scrutiny will continue to be shone on many of the current procedures and practices in EMS. Only in this way will the most cost-effective care be rendered for the greatest benefit of the largest number of citizens.

A diplomate of BCEM, Dr. Lemonick was originally trained in cardiothoracic surgery. He has practiced emergency medicine for almost 20 years, and currently is director of the emergency department at Highlands Hospital, near Pittsburgh, PA. His research interests include neurological and soft tissue infections, wound care, and biological terrorism.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The author has stated that no such relationships exist.

References

1. Callaham M. Quantifying the scanty science of prehospital emergency care. Ann Emerg Med 1997; 30(6):785-790.
2. Bledsoe BE. EMS myth #6. Air medical helicopters save lives and are cost-effective. Emerg Med Serv. 2003 Aug; 32(8):88-90.
3. Cowley RA. Trauma center: A new concept for the delivery of critical care. J Med Soc NJ 74:979–987, 1977.
4. Lerner EB, Moscati RM. The Golden Hour: Scientific Fact or Medical "Urban Legend"? Acad Emerg Med: 2001(8)7:758-760.
5. Shatney CH, Homan SJ, Sherck JP, Ho CC. The utility of helicopter transport of trauma patients from the injury scene in an urban trauma system. J Trauma 2002;53:817.
6. Reenstra WR, Tracy J, Hirsch E, Millham F. Evaluation of the "appropriateness" of triage requests for air transport to Level I trauma centers directly from the scene versus a community hospital. Ann Emerg Med 34(4):S73, 1999.
7. Bledsoe BE, Wesley AK, Eckstein M, Dunn TM, O'Keefe MF. Helicopter scene transport of trauma patients with nonlif.
8. Dula DJ, Plys K, Leicht M, et al. Helicopter versus ambulance transport of patients with penetrating trauma. Ann Emerg Med 2000. 36(4):S76.
9. Oppe S, De Charro FT. The effect of medical care by a helicopter trauma team on the probability of survival and the quality of life of hospitalized victims. Accid Anal Prev 2001;33(1):129-38.
10. Cocanour CS, Fisher RP, Ursic CM. Are scene flights for penetrating trauma justified? J Trauma 1997;43(1):83-6;discussion 86-8.
11. Thomas SH, Biddinger PD. Helicopter trauma transport: an overview of recent outcomes and triage literature. 2003. Curr Opin Anaesthesiol. Apr;16(2):153-8.
12. Chappell VL, Mileski WJ, Wolfe SE, et al. Impact of discontinuation of a hospital-based air ambulance service on trauma patient outcomes, J Trauma 2002;52:486-491.
13. Schiller WR, Knox R, Zinnecker H, et al. Effect of helicopter transport of trauma victims on survival in an urban trauma center. J Trauma 28(8):L1127–1134, 1988.
14. Thomas SH. Controversies in prehospital care: air medical response. 2005. Emergency Medicine Practice. 7;6. Periodical online. Available at: http://ebmedicine.net/topics. Accessed December 23, 2007.
15. Wong TW, Lau CC. Profile and outcomes of patients transported to an accident and emergency department by helicopter: Prospective case series. Hong Kong Med J 6(3):249–253, 2000.
16. Hotvedt R, Kristiansen IS, Forde OH, et al. Which groups of patients benefit from helicopter evacuation? Lancet 347:1362–1366, 1996.
17. Brazier J, Nicholl J, Snooks H. The cost and effectiveness of the London Helicopter Emergency Medical Service. J Health Serv Res Policy 1(4):232–237, 1996.
18. Eckstein M; Jantos T, Kelly N, et al. Helicopter transport of pediatric trauma patients in an urban setting: a critical analysis. 2002. J Trauma. 2002;53:340-344.
19. Moront ML, Gotschall CS, Eichelberger MR. Helicopter transport of injured children: System effectiveness and triage criteria. 1996J Pediatr Surg 31(8);183-6.
20. Tortella BJ, Sambol J, Lavery RF, et al. A comparison of pediatric and adult trauma patients transported by helicopter and ground EMS: managed-care considerations. 1996. Air Med J. Jan-Mar;15(1):24-8.
21. Branas CC, MacKenzie EJ, Williams JC, et al. Access to trauma centers in the United JAMA. 2005 Jun 1;293(21):2626-33.
22. Mallon WK. Trauma review. 2006. Audio-Digest Emergency Medicine. 23(11).
23. Stone CK, Thomas SH. Air medical transport. In: Tintinalli JE, Kelen GD., Stapczynski SJ (Eds). Emergency Medicine: A Comprehensive Study Guide 6th edition 2004. McGraw-Hill. p.11-15.
24. Gearhart PA, Wuerz R, Localio AR. Cost-effectiveness analysis of helicopter EMS for trauma patients.Ann Emerg Med. 1997 Oct;30(4):500-6. 28.
25. Bledsoe BE, Smith MG.Medical helicopter accidents in the United States: a 10-year review.J Trauma. 2004 Jun;56(6):1325-8.
26. Baker SP, Grabowski JG, Dodd RS, et al. EMS helicopter crashes: what influences fatal outcome? 2006 Ann Emerg Med. Apr;47(4):351-6.
27. Derzy M, Hustuit J, Boschert G, Wish J. Results and recommendations from the helicopter EMS pilot safety survey 2005. Air Med J. 2007 Jan-Feb;26(1):38-44.
28. Cunningham P, Rutledge R, Baker CC, Clancy TV.A comparison of the association of helicopter and ground ambulance transport with the outcome of injury in trauma patients transported from the scene. J Trauma. 1998 Jun;44(6):1114-5.
29. Brathwaite CE, Rosko M, McDowell R. et al. A critical analysis of on-scene helicopter transport on survival in a statewide trauma system. J Trauma. 1998 Jul;45(1):140-4;
30. Mayer T. Helicopter EMS. ACEP Scientific assembly. Washington, D.C. September 26-29. 2005
31. Diaz MA, Hendey GW, Winters RC. How far is that by air? The derivation of an air: ground coefficient. 2003. J Emerg Med. Feb;24(2):199-202.
32. Isakov AP. Souls on board: helicopter emergency medical services and safety. Ann Emerg Med. 2006 Apr;47(4):357-60.
33. Meier B, Saul S. Fatal crashes provoke debate on safety of sky ambulances. New York Times. February 28, 2005.
34. Meier B. As medical airlifts proliferate, the public price tag is rising. New York Times. May 3, 2005.
35. Thomas SH, Harrison TH, Buras WR, et al. Helicopter transport and blunt trauma mortality: a multicenter trial. J Trauma. 2002 Jan;52(1):136-45.
36. Kupas DF, Dula DJ, Pino BJ. Patient outcome using medical protocol to limit "lights and siren transport. Prehosp Diast Med. 1994:9(4).
37. Ho J, Casey B. Time saved with use of warning lights and sirens during response t requests for emergency medical aid in and urban environment. Ann Emerg Med 1998. 32(5);585-588.
38. Brown LH, Whitney CL, Hunt RC et al. Do warning lights and sirens reduce ambulance response times? Prehosp emerg care 2000. 4(1);70-74.
39. Hunt RC, Brown LH, Cabinum ES et al. Is ambulance transport time with lights and siren faster than that without? Ann Emerg Med 1995 Jun;25(6):85.

40. Wydro GC, Kraus LK, Yeh EC et al. Utilization of emergency lights and sirens by urban paramedics: analysis of indications for their use. Research forum abstract #257. ACEP Sci Ass'y. 2007. Ann Emerg Med. 50(3);S81.
41. Lacher ME, Bausher JC. Lights and siren in pediatric 911 ambulance transports: are they being misused? Ann Emerg Med. 1997 Feb;29(2):223-7.
42. Brown AL, Mann NC, Daya M,et al; for the Rapid Early Action for Coronary Treatment (REACT) Demographic, Belief, and Situational Factors Influencing the Decision to Utilize Emergency Medical Services Among Chest Pain Patients 2000 Circulation.;102:173.
43. Bledsoe BE. Emergency EMS mythology, Part 4. Lights and sirens save a significant amount of travel time and save lives. Emerg Med Serv. 2003 Jun; 32(6):72-3.
44. Ambulance crash-related injuries among emergency medical services workers- United States, 1991-2002. MMWR Morb Mort Wkly Rep 2003. 52(8):154-156.
45. Custalow CB, Gravitz CS. Prehosp Emerg Care. Emergency medical vehicle collisions and potential for preventive intervention. 2004 Apr-Jun;8(2):175-84.
46. Maguire BJ, Hunting KL, Smith GS, Levick NR. Occupational fatalities in emergency medical services: A hidden crisis. 2002. Annals of Emergency Medicine 40(6):625–632.
47. Clawson J, Martin R, Cady J, Maio R. The wake effect-emergency vehicle-related collisions. Prehospital Disaster Med. October-December 1997;12:274-277
48. Whiting JD, Dunn K, March JA, et al. Prehosp Emerg Care. EMT knowledge of ambulance traffic laws.1998 Apr-Jun;2(2):136-40.
49. Use of Warning Lights and Siren in Emergency Medical Vehicle Response and Patient Transport available at: http://www.naemsp.org/position.html; accessed December 30,2007.
50. Whiting JD, Dunn K, March JA, Brown LH. EMT knowledge of ambulance traffic laws. Prehosp Emerg Care. 1998 Apr-Jun;2(2):136-40.
51. Ducharme J.2005 Emerg Med Clin NA.. May 23 (2); 467-75. The future of pain management in emergency medicine.
52. Vassiliadis J, Hitos K, Hill CT. Factors influencing prehospital and emergency department analgesia administration to patients with femoral neck fractures. Emerg Med (Fremantle). 2002 Sep;14(3):261-6.
53. White LJ, Cooper JD, Chambers RM, Gradisek RE. Prehospital use of analgesia for suspected extremity fractures.Prehosp Emerg Care. 2000 Jul-Sep;4(3):205-8.
54. McEachin CC, McDermott JT, Swor R. Few emergency medical services patients with lower-extremity fractures receive prehospital analgesia. Prehosp Emerg Care. 2002 Oct-Dec;6(4):406.
55. Abbuhl FB, Reed DB. Time to analgesia for patients with painful extremity injuries transported to the emergency department by ambulance. Prehosp Emerg Care. 2003 Oct-Dec;7(4):445.
56. Kanowitz A, Dunn TM, Kanowitz EM, Dunn WW, Vanbuskirk K. Safety and effectiveness of fentanyl administration for prehospital pain management. Prehosp Emerg Care. 2006 Jan-Mar;10(1):1-7.
57. Galinski M, Dolveck F, Borron SW, Tual L, Van Laer V, Lardeur JY, Lapostolle F, Adnet F. A randomized, double-blind study comparing morphine with fentanyl in prehospital analgesia.Am J Emerg Med. 2005 Mar;23(2):114-9.
58. McManus JG Jr, Sallee DR Jr Pain management in the prehospital environment Emerg Med Clin North Am. 2005 May;23(2):415-31.
59. Todd KH, Samaroo N, Hoffman JR. Ethnicity as a risk factor for inadequate emergency department analgesia. JAMA. 1993;269(10):1537-9.
60. Todd KH, Deaton C, D'Adamo AP, Goe L. Ethnicity and analgesic practice. Ann Emerg Med. 2000;35(1):11-16.
61. Hennes H, Kim MK, Pirrallo RG. Prehospital pain management: a comparison of providers' perceptions and practices. Prehosp Emerg Care. 2005 Jan-Mar;9(1):32-9.
62. Swor R, McEachin CM, Seguin D, Grall KH. Prehospital pain management in children suffering traumatic injury. Prehosp Emerg Care. 2005 Jan-Mar;9(1):40-3.
63. Michael GE, Sporer KA, Youngblood GM.Women are less likely than men to receive prehospital analgesia for isolated extremity injuries. Am J Emerg Med. 2007 Oct;25(8):901-6.
64. Fosnocht DE, Swanson ER, Barton ED.Changing attitudes about pain and pain control in emergency medicine. Emerg Med Clin North Am. 2005 May;23(2):297-306.
65. Fullerton-Gleason L, Crandall C, Sklar DP Prehospital administration of morphine for isolated extremity injuries: a change in protocol reduces time to medication. Prehosp Emerg Care 2002 Oct-Dec; 6(4):411.
66. Pointer JE, Harlan K.Impact of liberalization of protocols for the use of morphine sulfate in an urban emergency medical services system.Prehosp Emerg Care. 2005 Oct-Dec;9(4):377-81.
67. Bledsoe B, Braude D, Dailey MW, Myers J, Richards M, Wesley K Simplifying prehospital analgesia. Why certain medications should or should not be used for pain management in the field. Prehosp Emerg Care. 2005 Jan-Mar;9(1):40-3.
68. Thomas SH, Rago O, Harrison T, Biddinger PD, Wedel SK. Fentanyl trauma analgesia use in air medical scene transports. J Emerg Med. 2005 Aug;29(2):179-87.
69. Bledsoe B, Braude D, Dailey MW, Myers J, Richards M, Wesley K Simplifying prehospital analgesia. Why certain medications should or should not be used for pain management in the field. JEMS. 2005 Jul;30(7):56-63.Links.
70. Svenson JE, Abernathy MK Ketamine for prehospital use: new look at an old drug 2007. American Journal of Emergency Medicine25, 977–980.
71. Alexander K, Scheck T, Greher M, et al. Prehospital Analgesia with Acupressure in Victims of Minor Trauma: A Prospective, Randomized, Double-BlindedTrial. 2002. Anesth Analg;95:723-727.
72. Lang T, Hager H, Funovits V, Barker R, Steinlechner B, Hoerauf K, Kober A.Prehospital analgesia with acupressure at the Baihui and Hegu points in patients with radial fractures: a prospective, randomized, double-blind trial.Am J Emerg Med. 2007 Oct;25(8):887-93.
73. Zhang NJ, Romig L, Barnard J. Evaluation of A New Prehospital Pain Management Protocol: Experiences From EMS Providers.2008 American Public Health Association 2008 Annual Meeting San Diego, CA October 25-29.
74. Alonso-Serra HM, Wesley K. for the National Association of EMS Physicians Standards and Clinical Practices Committee of the NAEMSP. Prehospital pain management position paper. Prehospital emergency care. 2003. vol 7, number 4. pp 482-8.
75. William T. Zempsky, MD; Joseph P. Cravero, MD; and the Committee on Pediatric Emergency Medicine and Section on Anesthesiology and Pain Medicine. Relief of Pain and Anxiety in Pediatric Patients in Emergency Medical Systems PEDIATRICS Vol. 114 No. 5 November 2004.
76. Barbara G. Lock, Eric Nazziola, Zhezhen Jin, Tracy Y. Allen, Peter S. Dayan, and Peter C. Wyer Opiate Analgesia Makes No Difference in the Diagnostic Accuracy of Undifferentiated Acute Abdominal Pain in the Emergency Department: A Meta-analysis. 2004. Acad Emerg Med 2004 11: 495.
77. Reynolds CM, Suber F, Curtis KM, et al. A Novel Pain Management Protocol Results in More Rapid Analgesia for Trauma Patients. 2004. Acad Emerg Med Volume 11, Issue5 497,
78. Lord BA, Parsell B.Measurement of pain in the prehospital setting using a visual analogue scale. Prehosp Disaster Med. 2003 Oct-Dec;18(4):353-8.
79. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. Ann EmergMed. 2001; 37:32-37.
80. Jemmett ME, Kendal KM, Fourre MW, et al. Unrecognized misplacement of endotracheal tubes in a mixed urban to rural emergency medical services setting. Acad Emerg Med. 2003; 10: 961-965.
81. Wang HE, Peitzman AB, Cassidy LD, et al. Out-of hospital endotracheal

intubation and outcome after traumatic brain injury. Ann Emerg Med 2004; 44:439-50.

82. Shafi, Shahid MD, MPH; Gentilello, Larry MD Pre-Hospital Endotracheal Intubation and Positive Pressure Ventilation Is Associated with Hypotension and Decreased Survival in Hypovolemic Trauma Patients: An Analysis of the National Trauma Data Bank. Journal of Trauma-Injury Infection & Critical Care. 59(5):1140-1147, November 2005.
83. Sen A, Nichani R.Best evidence topic report. Prehospital endotracheal intubation in adult major trauma patients with head injury. Emerg Med J. 2005 Dec;22(12):887-9.
84. Bochichhio GV, Ilahi O, Joshi M, Bochicchio K, Scalea TM. Endotracheal Intubation in the field does not improve outcome in trauma patients who present without an acutely lethal traumatic brain injury. Journal of Trauma, Injury, Infection and Critical Care 2003;54:307-311.
85. Gausche M, Lewis RJ, Stratton SJ, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. JAMA 2000; 283:783-90.
86. Dunford JV, Davis DP, Ochs M, et al. Incidence of transient hypoxia and pulse rate reactivity during paramedic rapid sequence intubation. Ann Emerg Med. 2003;42:721- 728.
87. Wang HE, Yealy DM. How many attempts are required to accomplish out-of-hospital endotracheal intubation? Academic Emergency Medicine 2006 Apr;13(4):372-7.
88. Davis DP, Peay J, Sise MJ,et al. The impact of prehospital endotracheal intubation on outcome in moderate to severe traumatic brain injury.J Trauma. 2005 May;58(5):933-9.
89. Jones JH, Murphy MP, Dickson RL. Emergency physician-verified out-of-hospital intubation: miss rates by paramedics. Academic Emergency Medicine 2004 Jun;11(6):707-9.
90. Ufberg JW, Bushra JS, Patel D, et al. A new pepsin assay to detect pulmonary aspiration of gastric contents among newly intubated patients. Am J Emerg Med. 2004 Nov;22(7):612-4.
91. Ufberg JW, Bushra JS, Karras DJ, et al. Aspiration of gastric contents: association with prehospital intubation. Am J Emerg Med. 2005 May;23(3):379-82.
92. Silvestri S, Ralls GA, Krauss B, et al. The effectiveness of out-of hospital use of continuous end-tidal carbon dioxide monitoring on the rate of unrecognized misplaced intubation within a regional emergency medical services system. Ann Emerg Med. 2005;45:497-503.
93. George S, Macnab AJ.Evaluation of a semi-quantitative CO2 monitor with pulse oximetry for prehospital endotracheal tube placement and management. Prehosp Disaster Med. 2002 Jan-Mar;17(1):38-41.
94. Hendey GW, Shubert GS, Shalit M, Hogue B. The esophageal detector bulb in the aeromedical setting. J Emerg Med. 2002 Jul;23(1):51-5.
95. Wang VJ, Krauss B. Carbon dioxide monitoring in emergency medicine training programs. Pediatr Emerg Care. 2002 Aug;18(4):251-3.
96. Tanigawa K, Shigematsu A. Choice of airway devices for 12,020 cases of nontraumatic cardiac arrest in Japan.Prehosp Emerg Care. 1998 Apr-Jun;2(2):96-100.
97. Tanigawa K, Takeda T, Goto E, Tanaka K. Accuracy and reliability of the self-inflating bulb to verify tracheal intubation in out-of-hospital cardiac arrest patients. Anesthesiology. 2000 Dec;93(6):1432-6.
98. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Circulation. 2005;112 [Suppl I]:IV-1-IV-5.
99. Yannopoulos D, Aufderheide TP.Use of the Impedance Threshold Device (ITD). Resuscitation. 2007 Oct;75(1):192-3; author reply 193-4.
100. Pirracchio R, Payen D, Plaisance P. The impedance threshold valve for adult cardiopulmonary resuscitation: a review of the literature. Curr Opin Crit Care. 2007 Jun;13(3):280-6.
101. Menegazzi JJ, Salcido DD, Menegazzi MT. Effects of an impedance threshold device on hemodynamics and restoration of spontaneous circulation in prolonged porcine ventricular fibrillation. Prehosp Emerg Care. 2007 Apr-Jun;11(2):179-85.
102. Aufderheide TP, Pirrallo RG, et al. Clinical evaluation of an inspiratory impedance threshold device during standard cardiopulmonary resuscitation in patients with out-of-hospital cardiac arrest. Crit Care Med. 2005 Apr;33(4):734-40.
103. Pirrallo RG, Aufderheide TP, et al. Effect of an impedance threshold device on hemodynamics during conventional manual cardiopulmonary resuscitation. Resuscitation 2005; 66:13-20.
104. Wolcke BB, Mauer DK, et al. Comparison of standard CPR versus the combination of active compression-decompression cardiopulmonary resuscitation and an inspiratory impedance threshold device for out-of-hospital cardiac arrest. Circulation 2003; 108(18):2201-5.
105. Plaisance P, Lurie KG, Payen D. Inspiratory impedance during active compression-decompression CPR: a randomized evaluation in patients in cardiac arrest. Circulation 2000;101(9):989-94.
106. Plaisance P, Lurie KG, et al. Evaluation of an impedance threshold device in patients receiving active compression-decompression cardiopulmonary resuscitation for out of hospital cardiac arrest. Resuscitation 2004;61(3):265-71.
107. Plaisance P, Soleil C, et al. Use of an inspiratory impedance threshold device on a facemask and endotracheal tube to reduce intrathoracic pressures during the decompression phase of active compression decompression cardiopulmonary resuscitation. Crit Care Med 2005; 33(5):990-4.
108. Galinski M, Tréoux V, Garrigue B et al. Intracuff pressures of endotracheal tubes in the management of airway emergencies: the need for pressure monitoring. Ann Emerg Med. 2006 Jun;47(6):545-7.
109. Svenson JE, Lindsay MB, O'Connor JE.Endotracheal intracuff pressures in the ED and prehospital setting: is there a problem? Am J Emerg Med. 2007 Jan;25(1):53-6.
110. Guyette FX, Wang H, Cole JS. King airway use by air medical providers. Prehosp Emerg Care. 2007 Oct-Dec;11(4):473-6.
111. Smally AAANA J. The esophageal-tracheal double-lumen airway: rescue for the difficult airway.2007 Apr;75(2):129-34.
112. Gausche-Hill M. Ensuring quality in prehospital airway management. Curr Opin Anaesthesiol. 2003 Apr;16(2):173-81.
113. Zheng ZJ, Croft JB, Giles WH, Mensah GA.Sudden cardiac death in the United States, 1989 to 1998. Circulation. 2001 Oct 30;104(18):2158-63.
114. Stiell IG, Wells GA, Field B, et al. for the OPALS Study Group Advanced cardiac life support in out-of-hospital cardiac arrest. N Engl J Med 2004; 351: 647-56.
115. Stiell IG, Spaite DW, Field B, et al. for the OPALS Study Group. Advanced life support for out-of-hospital respiratory distress. N Engl J Med. 2007 May 24; 356(21):2156-64.
116. Pitetti R, Glustein JZ, Bhende MS.Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care. 2002 Jul-Sep;6(3):283-90.
117. Valenzuela TD, Roe DJ, Nichol G, Clark LL, Spaite DW, Hardman RG.: Outcomes of rapid defibrillation by security officers after cardiac arrest in casinos.N Engl J Med. 2000 Oct 26;343(17):1206-9.
118. Kaluski E, Uriel N, Milo O, et al .Management of cardiac arrest in 2005: an update. Isr Med Assoc J. 2005 Sep;7(9):589-94.
119. De Maio VJ, Stiell IG, Wells GA et al. for the OPALS study group. Optimal defibrillation response intervals for maximum out-of-hospital cardiac arrest survival. Ann Emerg Med 2003; 42(2):242-250.
120. SOS-KANTO study group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. Lancet. 2007 Mar 17;369(9565):920-6.
121. Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation for patients with out-of-hospital ventricular fibrillation. JAMA. 2003;289:1389-1395.
122. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary

resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation; a randomized trial. JAMA 1999;289(13):1182-8.

123. Wik L, Kramer-Johansen J, Myklebust H et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA. 2005 Jan 19;293(3):299-304.

124. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA. 2005 Jan 19;293(3):305-10.

125. Pons PT, Haukoos JS, Bludworth W, et al. Paramedic Response Time: Does It Affect Patient Survival? 2005. Acad Emerg Med Volume 12, Issue7 594-600.

126. Blackwell TH, Kaufman JS. Response Time Effectiveness Comparison of Response Time and Survival in an Urban Emergency Medical Services System 2002. Acad Emerg Med.Volume 9, Issue4 288-295.

127. Fitch J. Response times: myths, measurement & management. JEMS.2005. Sep;30:47-56.

128. Cummins RO, Eisenberg MS, Letwin PE , et al. Automatic external defibrillators used by emergency medical technicians: A controlled clinical trial. JAMA. 1987 ;257:1605 -1610.

129. Myerburg RJ, Fenster J, Velez M, et al. Impact of Community-Wide Police Car Deployment of Automated External Defibrillators on Survival From Out-of-Hospital Cardiac Arrest. Circulation 2002; 106(9): 1058-64.

130. Hallstrom AP, Ornato JP, Weisfeldt M, et al. Public Access Defibrillation Trial Investigators. Public-access defibrillation and survival after out-of-hospital cardiac arrest. N Engl J Med. 2004 Aug 12;351(7):637-46.

131. Stotz M, Albrecht R, Zwicker G, et al. EMS defibrillation-first policy may not improve outcome in out-of-hospital cardiac arrest. Resuscitation. 2003 Sep;58 (3):277-82.

132. Papson K and Mosesso VN Jr. Ten years of police defibrillation: Program characteristics and personnel attitudes. Prehosp Emerg Care 2005 Apr/Jun; 9:186-90.

133. Groh WJ, Newman MM., Beal PE, et al. Limited response to cardiac arrest by police equipped with automated external defibrillators lack of survival benefit in suburban and rural Indiana—the police as responder automated defibrillation evaluation (PARADE) 2001 Academic Emergency Medicine Volume 8, Number 4 324-330.

134. Forrer CS, Swor RA, Jackson RE, et al. Estimated cost effectiveness of a police automated external defibrillator program in a suburban community: - 7 years experience. 2002; Resuscitation, Volume 52, Number 1, January, 23-29.

135. Pell JP, Sirel JM, Marsden AK, et al. Potential impact of public access defibrillators on survival after out of hospital cardiopulmonary arrest: retrospective cohort study BMJ 2002;325:515 (7 September).

136. Pell, JP; Walker, A; Cobbe, SM. Cost-effectiveness of automated external defibrillators in public places: con. Current Opinion in Cardiology. 22(1):5-10, January 2007.

137. Eisenberg M. Dissemination of defibrillators—medical vs consumer scenarios. JAMA. 2000;284(11):1435–1438).

138. Gold LS, Eisenberg M. Cost-effectiveness of automated external defibrillators in public places: pro. Curr Opin Cardiol. 2007 Jan ;22 (1):1-4 17143037.

MEDICAL ETHICS
WITHOUT THE RHETORIC

The cases presented here involve real physicians and patients. Unlike the cases in medical ethics textbooks, these seldom involve cloning, bizarre treatments, or stem cell research. We focus on cases common to the practice of medicine in a variety of contexts.

Mark Pastin, Ph.D.

I am considered an expert in medical ethics and I have the Harvard Ph.D. and academic history to prove it. But the only thing I really know about medical ethics is that there are no experts or recipes. The majority of cases are circumstantially unique and require the viewpoints of the practitioners and patients involved. For this reason, I am soliciting your input at mpastin@healthethicstrust.com on the cases discussed here. Reader perspectives along with my own viewpoint will be published in the issue following each case presentation. Of course, we are also interested in cases that readers wish to submit for consideration.

CASE ONE
PUT THE PATIENT FIRST?

Your patient is a pregnant 12-year-old girl and you are discussing options with her and her parents, who are practicing Catholics. The girl is pregnant by her boyfriend so there is no question of molestation. The girl is in poor health and, even putting aside her age, continuing the pregnancy is not medically advisable. The parents and the girl do not want to continue the pregnancy but have a special request. The request is that the girl's medical record not state that she had an abortion. In your mind, you know you might record the procedure as a D&C with product, as any physician who reads the record will understand it. You ask yourself, "Isn't the only difference between a D&C and an abortion what is in the mind of the patient? Is that any of my business?" The family will pay for the procedure out of pocket no matter what it is called. You ponder whether or not to honor their wish.

This is a situation I have seen many times, particularly in EDs and practices affiliated with religious systems that prohibit abortion. Of course, there could be any number of extenuating circumstances and additional details. But please address the case on the basis of the information provided as best you can. There will be an analysis of this case along with a new case in the next issue.

Your input is requested. Email your responses to: mpastin@healthethicstrust.com

Lyme Disease and Rocky Mountain Spotted Fever:
Diagnosis, Prevention, and Management

Ribhi Hazin, M.D.
Jamil Y. Abuzetun, M.D.
Manar Suker, M.D.

ABSTRACT

Most reported cases of Lyme disease and RMSF occur in the spring and summer months when tick infestations reach their peak. The primary care physician can play an important role during peak periods by remaining vigilant to early signs of both Lyme disease and RMSF. Although both Lyme disease and RMSF are treatable, clinicians should be aware of early signs of both diseases to help reduce long-term complications. Patients should also be encouraged to adopt personal protective measures to minimize exposure to ticks and, thus, either of these diseases. The following is a comprehensive overview in which pertinent diagnostic aspects of each disease are highlighted with a particular emphasis placed upon the role of the primary care physician in treating and minimizing the risks of Lyme disease and RMSF.

Key Words: Lyme Disease, Rocky Mountain Spotted Fever, Rickettsia rickettsii, American dog tick, Dermacentor variabilis, Ixodes tick, Borrelia burgdorferi, spirochete, tick-borne disease

Rocky Mountain Spotted Fever (RMSF)

Incidence

3.8 cases per 1 million persons in 2002.[1,2] Over 90% of patients with Rocky Mountain spotted fever are infected during summer months (Table 1).[1] Is most common in the Southeastern United States.[1]

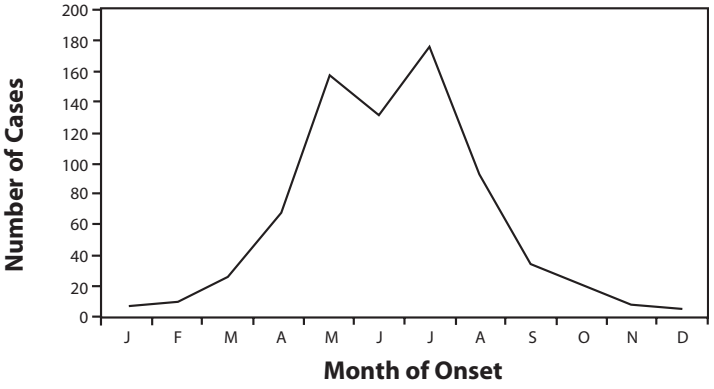
Mortality

The overall mortality rate is currently 1.4% [2]; however, the case-fatality rate can be as high as 4.8% in children under the age of 5.[2]

Discussion

Rocky Mountain spotted fever (RMSF), the most severe of the rickettsioses, is a serious tick-borne disease that is endemic in the United States.[3] It is caused by R. rickettsii and is transmitted by Dermacentor andersoni (wood tick) or Dermacentor variabilis (dog tick).[4] Although it is most commonly known as a cause of fever and rash, it can have systemic manifestations.[3] The initial presentation of RMSF is often nonspecific and may resemble many other infectious and non-infectious diseases and can represent a diagnostic challenge.[4] The classic triad for the disease is rash, fever, and history of being bitten by a tick. Unfortunately, these findings are present in less than half of cases and only in the early clinical presentation.[2,4] Most patients will complain of fever (98%), rash (97%), nausea and/or vomiting (73%), and headache (61%), but can also present with no sign other than the rash (>50%).[5] Furthermore, although the dog tick is responsible for most cases of RMSF, less than half of those infected report antecedent tick bites.[5]

Table 1: Monthly Case Distribution of RMSF in 2002[1]



The rash seen in RMSF is characteristically macular, spotted (petechial), and begins on the ankles and wrists and then generalizes throughout the body.[6] This, perhaps, is due to the fact that the clinical picture changes according to how long prior to admission the tick bite occurred.[6,7,8] For example, in patients presenting within the first two weeks after the tick bite, typical symptoms would be very elevated fever (often greater than 101), headaches, and constitutional symptoms such as myalgias, anorexia, diar-

rhea, or vomiting.[6,7] As a result of the nonspecific nature of such symptoms, patients living in endemic areas, who present with high fever and a history of tick bite, should be considered for the disease.[8] Between 60-80% of patients presenting two weeks or longer after the tick bite will have the characteristic rash along with the aforementioned nonspecific symptoms.[1,8]

Diagnosis

Laboratory confirmation of the diagnosis can be attained via immunofluorescence assay (IFA), complement fixation, latex agglutination, or immunodetection to detect bacterial antigens in a skin biopsy specimen.[2,8] Unfortunately, no rapid diagnostic test currently exists for RMSF and serologic confirmation of the disorder often requires serum obtained at least two weeks after onset of symptoms.[2] Furthermore, culture of Rickettsia can only be done via specialized laboratories and often requires two weeks for confirmatory results.[2,9] However, the rapid progression of the disease and the associated mortality make empiric therapy a standard of therapy even in the absence of serologic confirmation.[8,9] Delays in diagnosis and initiation of appropriate therapy are unacceptably common in RMSF, and, as a result, patient outcomes are often suboptimal.[2,5] This is particularly troubling since up to 14% of infected individuals will develop neurologic deficits.[5]

The lack of knowledge of RMSF among clinicians can be responsible for the diagnostic shortcomings. For instance, a recent study demonstrated that only 21% of family practice physicians and 25% of emergency medicine physicians surveyed were able to correctly identify the drug of choice for treating RMSF.[8] Continuing efforts to educate first responders and primary care physicians on the intricacies of the disorder should focus on appropriate selection of antibiotics and prompt initiation of empiric prior to the onset of rash in suspected patients.[8,9]

Treatment

The current treatment of choice for RMSF is 200 mg of doxycycline b.i.d until three days after resolution of fever.[2,10] Given the high mortality associated with the condition, it is recommended that doxycycline be prescribed empirically in all suspected cases.[9] The only exception is pregnant women for whom doxycycline should be avoided.[11] Oral chloramphenicol therapy remains the only effective alternative in treating pregnant women with RMSF.[2,8,11] All individuals suspected of the condition should receive prompt antimicrobial therapy to avoid potentially fatal complications.[9,11]

Lyme Disease

Incidence

There are 29.2 cases per 100,000 population with 93% of those cases occurring among residents in ten states, mostly in the northeastern United States.[12]

Mortality

Lyme disease is rarely fatal.

Discussion

Lyme disease is a multisystem inflammatory condition caused by the organism Borellia burgdorferi, which is transmitted to humans by the bite of infected Ixodes ticks.[12] The disease exists in three distinct clinical stages, but clinical features and symptoms of stages may overlap.[13] Stage I describes early localize Lyme disease, whose common features include flu-like symptoms, chills, headaches along with erythema migrans (EM) or a circular red rash that tends to generalize from the site of the tick bite.[12,13] Although EM characteristically exists as a solitary lesion, in up to one-fourth of patients, multiple annular lesions may also develop. Stage II or early disseminated Lyme disease describes the clinical features of the disorder several weeks to months after being infected.[14] Characteristic clinical features of Stage II include intermittent pain and swelling in the joints, especially the knees, weakness in the extremities as a result of neurologic deficits, and arrythmias.[15-17] Stage III or late persistent Lyme disease is often accompanied by chronic arthritis, numbness and weakness in the limbs, and pericarditis.[15-18] Increased risk of chronic neurologic and cardiac complications accompanies the onset of Stage III of the disease.[17-18]

Diagnosis

Recognition of salient clinical signs and accompanying history of exposure are important to accurately diagnose the condition. The current diagnostic paradigm recommended is utilization of immunosorbet assay (ELISA) or immunoblotting, two techniques considered essential for diagnosis.[16,19] Although it can be confirmatory, culturing B. burgdorferi from patient serum or skin specimens has proven largely ineffective.[20,21] Assessing serum for specific antibodies against B. burgdorferi can be an effective adjunctive diagnostic test, especially four to six weeks after onset of symptoms when such antibodies peak in number.[20,21]

Treatment

Since the preponderance of patients with early Lyme disease can be cured with the appropriate therapy, the major goal of therapy in the disease is to eliminate the causative organism.[22] Lyme disease tends to be most responsive to therapy, particularly when treatment is initiated early in the course of the disease. The treatment of choice for the disorder consists of oral doxycycline 100 mg b.i.d for 21 days or oral amoxicillin 500 mg t.i.d for 21 days.[15,22] Treating patients in later stages of Lyme can pose a challenge. However, treatment of Lyme-induced meningitis with intravenous penicillin G has demonstrated promising results.[15,16] Further, Lyme-induced arthritis has been successfully treated with oral antibiotics in the past.[16,23]

Prevention

Contrary to popular belief, both Lyme disease and RMSF can occur throughout the country. As with any tick-borne disease, all patients, especially those living in endemic areas, should be encouraged to adopt the following protective measures:[24-28]

1) Avoid sandals or open-toe shoes;
2) Wear light-colored garments for easier identification of ticks;
3) Spray tick repellant on exposed skin and over clothing;
4) Wear long-sleeve shirts and long pants whenever possible;
5) Tuck shirt into pants and pants into socks to prevent ticks from reaching exposed skin;
6) Wear a hat;
7) Check body for ticks at least once a day (especially in inguinal regions, on the scalp, and behind the neck);
8) Avoid tick-infested areas, especially during peak months (spring/summer);
9) Wash and dry clothing in high temperature in order to destroy lingering ticks;
10) Clear vines, leaves, or woodpiles around the home, as they can provide shelter for ticks.

Conclusions

Although both RMSF and Lyme disease tend to be most common to specific geographic locales, both diseases can occur throughout the United States. Since the spring and summer months coincide with peak infestation of the ticks responsible for the two diseases, clinical awareness coupled with clinical preparedness can be instrumental in early recognition of these two diseases. Early diagnosis of both RMSF and Lyme disease is critical, since untreated infection may cause irreversible damage to the central nervous and cardiovascular systems.

Ribhi Hazin, M.D., is a John F. Kennedy Fellow at Harvard University, where he is focusing on health policy and ethics.

Jamil Y. Abuzetun, M.D., is currently completing internal medicine residency training at Creighton University Medical Center in Omaha, NE. He was recently appointed Chief Resident, a position he will assume in July 2009.

Manar Suker, M.D., is a recent medical school graduate living in Omaha, NE. She is currently raising a family prior to beginning her internal medicine residency, scheduled for 2010.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist.

References

1. Centers for Disease Control and Prevention (CDC). Fatal cases of Rocky Mountain spotted fever in family clusters-three states, 2003. MMWR Morb Mortal Wkly Rep 2004;53:407-10.
2. Chapman AS, Murphy SM, Demma LJ, Holman RC, Curns AT, McQuiston JH, et al. Rocky Mountain spotted fever in the United States, 1997-2002. Vector Borne Zoonotic Dis 2006;6:170-8.
3. Doyle A, Bhalla KS, Jones JM 3rd, Ennis DM. Myocardial involvement in Rocky Mountain spotted fever: a case report and review. Am J Med Sci 2006;332:208-10.
4. Simser JA, Macaluso KR, Mulenga A, Azad AF. Immune-responsive lysozymes from hemocytes of the American dog tick, Dermacentor variabilis and an embryonic cell line of the Rocky Mountain wood tick, D. andersoni. Insect Biochem Mol Biol 2004;34:1235-46.
5. Buckingham SC, Marshall GS, Schutze GE, Woods CR, Jackson MA, Patterson LE, et al. Clinical and laboratory features, hospital course, and outcome of Rocky Mountain spotted fever in children. J Pediatr 2007;150:180-4, 184.e1.
6. Aber C, Alvarez Connelly E, Schachner LA. Fever and rash in a child: when to worry? Pediatr Ann 2007;36:30-8.
7. Randall MB, Walker DH. Rocky Mountain spotted fever. Gastrointestinal and pancreatic lesions and rickettsial infection. Arch Pathol Lab Med 1984;108:963-7.
8. O'Reilly M, Paddock C, Elchos B, Goddard J, Childs J, Currie M. Physician knowledge of the diagnosis and management of Rocky Mountain spotted fever: Mississippi, 2002. Ann N Y Acad Sci 2003;990:295-301.
9. Lacz NL, Schwartz RA, Kapila R. Rocky Mountain spotted fever. J Eur Acad Dermatol Venereol. 2006 Apr;20(4):411-7.
10. Consequences of delayed diagnosis of Rocky Mountain spotted fever in children--West Virginia, Michigan, Tennessee, and Oklahoma, May-July 2000. SO - MMWR Morb Mortal Wkly Rep 2000;49:885-8.
11. Herbert WN, Seeds JW, Koontz WL, Cefalo RC. Rocky Mountain spotted fever in pregnancy: differential diagnosis and treatment. South Med J 1982;75:1063-6.
12. Centers for Disease Control and Prevention (CDC). Lyme Disease-United States, 2003-2005. MMWR Morb. Mortal Wkly Rep 2007;56:573-576.
13. Stonehouse A, Studdiford JS, Henry CA. An Update on the Diagnosis and Treatment of Early Lyme Disease: "Focusing on the Bull's Eye, You May Miss the Mark" J Emerg Med. 2007 Oct 16; [Epub ahead of print].
14. Créange A. Clinical manifestations and epidemiological aspects leading to a diagnosis of Lyme borreliosis: neurological and psychiatric manifestations in the course of Lyme borreliosis. Med Mal Infect. 2007 Jul-Aug;37(7-8):532-9.
15. Hansmann Y. Treatment of Lyme borreliosis secondary and tertiary stages Med Mal Infect. 2007 Jul-Aug;37(7-8):479-86.
16. Shapiro ED. Lyme disease. Adv Exp Med Biol. 2008;609:185-95.
17. Vazquez M, Sparrow SS, Shapiro ED. Long-term neuropsychologic and health outcomes of children with facial nerve palsy attributable to Lyme disease. Pediatrics 2003;112:e93-7.
18. Shin JI, Lee JS. Complete heart block: Henoch-Schonlein purpura or Lyme carditis? Intern Med J 2007;37:69.
19. De Martino SJ. Role of biological assays in the diagnosis of Lyme borreliosis presentations. What are the techniques and which are currently available? Med Mal Infect 2007 Jul-Aug;37(7-8):496-506.
20. Assous MV. Laboratory methods for the diagnosis of clinical forms of Lyme borreliosis. Med Mal Infect 2007 Jul-Aug;37(7-8):487-95.
21. Marangoni A, Moroni A, Accardo S, Cevenini R. Borrelia burgdorferi VlsE antigen for the serological diagnosis of Lyme borreliosis. Eur J Clin Microbiol Infect Dis. 2008 Jan 16; [Epub ahead of print].
22. Monsel G, Canestri A, Caumes E. Antibiotherapy for early localized Lyme disease. Med Mal Infect 2007 Jul-Aug;37(7-8):463-72.

23. Klempner MS, Hu LT, Evans J, Schmid CH, Johnson GM, Trevino RP, et al. Two controlled trials of antibiotic treatment in patients with persistent symptoms and a history of Lyme disease. N Engl J Med 2001;345:85-92.

24. Piesman J, Eisen L. Prevention of Tick-Borne Diseases. Annu Rev Entomol 2008 Jan 7;53:323-343.

25. Nadelman RB, Wormser GP. Reinfection in patients with Lyme disease. Clin Infect Dis 2007;45:1032-8.

26. Zeller JL, Burke AE, Glass RM. JAMA patient page. Lyme disease. JAMA 2007;297:2664.

27. Wormser GP, Dattwyler RJ, Shapiro ED, Dumler JS, O'Connell S, Radolf JD, Nadelman RB. Single-dose prophylaxis against Lyme disease. Lancet Infect Dis 2007;7:371-3.

28. Boulanger N. What primary prevention should be used to prevent Lyme disease? Med Mal Infect 2007;37:456-62.

ABPS Certificate Frames Now Available

These beveled, concave, crystal frames are perfect for displaying your ABPS board certification certificate in a reception room or as an addition to your office.

Please allow four weeks for delivery since these are special orders.

Diplomates may purchase a color-copy, reduced size (5x7) ABPS certificate inserted into the crystal frame and a full-size, unframed ABPS certificate for $125.00. Additional crystal frames with inserted certificates may be purchased for $50.00 each.

Please note that framing for full-size ABPS certificates can be ordered through Framing Success at www.framingsuccess.com

Order Form

Yes, I would like to order

_____ Crystal frame with inserted reduced-size certificate, includes one full-size, unframed certificate ($125.00)

_____ Additional crystal frames with inserted reduced-size certificate ($50.00 each)

_____ Full-size, **unframed** certificate(s) ($85.00)

Shipping Address:

Name:_____ Address:_____

City:_____ State: _____ Zip:_____ Country: _____

Method of Payment: (Make checks payable to ABPS) ☐Visa ☐Mastercard ☐American Express

Credit Card # _____ Expiration Date: _____

Signature_____

American Board of Physician Specialties •5550 West Executive Drive • Suite 400 • Tampa, FL 33609 • 813-433-ABPS (2277) • Fax: 813-830-6599 • www.abpsus.org

An Approach to the Initial Care of Patients with Chest Pain in an Emergency Department Located in a Non-Cardiac Center

Alex A. Agostini-Miranda, M.D.
Loren A. Crown, M.D.

Abstract

Emergency department management of chest pain is a common, with potentially serious problem. With multiple risk factors, target groups, and care pathways, the myriad etiologies of chest pain can be perplexing. This article describes some of the diagnostic and risk stratification scenarios currently used in departments which do not have percutaneous cardiac intervention capabilities.

Introduction

Patients who appear in the emergency department, urgent care centers, or offices who state that their chief complaint is chest pain deserve an appropriate medical evaluation. In the US, there are approximately six to seven million visits per year to the emergency department or chest pain units due to a chief complaint of chest pain, of which 1.6 million are admitted with a diagnosis of acute coronary syndrome (ACS). Of these, 25 percent (400,000) meet criteria for acute myocardial infarction (AMI) by EKG criteria of ST elevation (STEMI), whereas 75 percent meet criteria for the diagnosis of unstable angina (UA) or non-ST elevation MI (NSTEMI).[1-3] This means that the majority of chest pain complaints are not due to ACS. Non-cardiac chest pain (NCCP) is the second most common reason for presentation to the ED and accounts for approximately two to five percent of all visits.[4-5]

Though it is obvious that only a minority of these patients have a coronary artery problem, the tragedy of a missed diagnosis and the resulting medico-legal problem, requires that we should routinely view chest pain initially as an acute coronary syndrome until proven otherwise. Four other critical causes of acute morbidity or mortality follow closely in the differential; they are: pulmonary embolism, aortic dissection, pneumothorax, and pneumonia.[6] Multiple other etiologies are possible and may need to be considered. It is only after an appropriate medical history, a physical exam, and investigational steps are completed that the physician will be able to exclude some of these entities. Often it takes multiple investigations over time to come to an adequate definition of the etiology of the chest pain. Nonetheless, screening for these critical causes of death are paramount for the emergency physician managing chest pain. Finally, remember, causes of chest pain are not limited to the thorax.

Causes of Chest Pain – The Short List!

Cardiovascular:
ACS/CAD/Angina
Anomalous left coronary
Aortic dissection
Aortic stenosis
Arrhythmias
Cardiac tamponade
Cardiomyopathy
Hypertrophic cardiomyopathy
Kawasaki disease
Mitral valve prolapse
Pericarditis/ myocarditis
Valvular heart disease

Drug abuse:
Amphetamine
Cocaine
"Diet pills"
Party Drugs (LSD and Ecstacy)

Gastric:
Diverticular disease
Esophageal spasm
Esophagitis
Gastritis
Gastroesophageal reflux disease

Miscellaneous:
Anemia (including sickle cell)
Herpes zoster
Thyroid/adrenal problems
Toxins

Musculoskeletal:
Arthritis
Costochondritis
Fibromyalgic
Muscular strain
Myofasciitis
Trauma

Psychological:
Somataform disorder
Anxiety/panic attacks
Depression

Respiratory/pulmonary:
Asthma/reactive airway disease
Cancer
Foreign Body
Hypoxia
Pleurisy
Pneumomediastinum
Pneumonia
Pneumothorax
Pulmonary embolism
Tracheitis

Chest Pain in Children

Widespread public information messages have made people more aware of heart disease in children, so it is not surprising to find many anxious parents camped out in the ED with a healthy, non-toxic child who has a complaint of chest pain. Some of this anxiety is based on media cases involving sudden cardiac death in young children. It should be remembered that in the majority of cases sudden death in children does not include complaints of chest pain. The etiology of sudden death in children has been postulated to be due to structural defects or to arrhythmias that could have its origin in genetic abnormalities of the ion channels that affect the channel structure and function.[7-8] Of all cases of chest pain reported in children only 5% are attributable to a cardiac etiology.[9]

As you begin the evaluation of a child with chest pain consider that in most cases there will be a child attached to two parents. When you remember that the worst nightmare for any parent is the loss of a child, make certain to pay heed to the fact that you may have three patients and not just one.

Start your evaluation by inspecting the vital signs (they are called vital for a reason and the emergency physician will be held responsible for any abnormal vital sign that remains unexplained) making sure to document the four cardinal ones, plus the O_2 saturation, as you observe the child. An active, playful child may be the best sign of reassurance that you, the doctor, can obtain, but it is rarely sufficient for the parent. Therefore, you must obtain the complete history of pain documenting the quality, intensity, location, temporal relationship to events, provocative and palliative factors, and associated symptoms. Trauma, including child abuse, must be sought and excluded. Look to the history and review of systems for weight loss, fatigue, headache, syncope, altered mental status, palpitations, pain on exertion, cough, wheezing, vomiting, and recent fevers/URIs that may be associated with myocarditis, athletic heart syndrome, or drug abuse.

Continue your evaluation by performing an appropriate physical exam, concentrating on the cardiovascular, respiratory, gastrointestinal, and musculoskeletal systems. Remember to document your findings ("if you didn't write it, you didn't do it"). The following tests could be performed in order to reassure yourself and the parents and to clarify doubts about the diagnosis: the chest x-ray (PA and lateral) and 12-lead EKG are usually useful, and additional hematologic and chemistry profiles may be appropriate for specific cases.

All chest pain associated with dyspnea, syncope, and palpitations, especially if it occurs during strenuous physical activity, will need to be more thoroughly evaluated and often results in a pediatric cardiologic consultation. But, in the vast majority of cases, at the end of your evaluation you will not find a definite cause for chest pain in children seen in the ED. Be honest with the parents, but always tell them if you do not find any of the serious causes of pain that are associated with acute morbidity or mortality. Consult as needed; follow up is almost always appropriate.

Chest Pain in Adults

Heart disease remains the leading cause of death in the United States with an estimated direct and indirect cost of approximately $142.5 billion for 2006,[4,10-11] but certainly not all people who arrive in the ED or clinic with the complaint of chest pain are experiencing heart disease. Certainly, as patients become older, more obese, and less active, the risk of developing coronary artery disease and dying from it increases. It is also important to remember that it is not only ACS (whether STEMI/NSTEMI) that kills patients, but other etiologies such as pulmonary embolisms, dissecting aortas, pneumothorax, and pneumonias that may also kill patients.

Pulmonary embolism is a legendary challenge for emergency physicians. The classic description of sudden onset of pleuritic chest pain associated with shortness of breath, abnormal vital signs, and hypoxia is not the most common presentation. Well's criteria is the most widely used method for evaluating patients by stratifying them into risk categories. Low-risk patients currently are being excluded by use of the d-dimer. All others require investigations by ultrasonography and/or CT angiography; various strategies exist for thrombolysis. Patients with aortic dissection are described as presenting with severe tearing or sharp chest pain that is sudden in onset. However, this classical description is unfortunately not always seen. Many patients present with neurological symptoms or syncope as the initial complaint. Hypertension in the elderly remains the main risk group, but those with historical findings such as connective tissue disorders and sudden death in family members are also frequently found in the high-risk group.

Patients with pneumonia should present with fever, cough, and infiltrates on the x-ray; however, it is altogether too common that they present with merely lack of energy, perception of being "sick," and myalgia, especially in the elderly. In addition, there are numerous other causes for chest pain (see Table 1) including patients with palpable discomfort on chest wall examination. Remember that up to 15% of acute MI patients may present with this finding and it certainly does not prevent them from having a concomitant coronary event.[12]

Scoring Risk in ACS

We are all aware of the "door to diagnosis/drug/transfer guidelines" set forth for patients who state that they have chest pain. At triage, most all hospitals have implemented protocols to stratify patients into high-, moderate-, and low-risk groups, but remember that protocols are not perfect. Our population has dramatically increased its incidence of the obese and morbidly obese (2/3 and 1/3 of the population respectively) and the number of patients with metabolic syndrome has burgeoned. The percentage of children afflicted has increased and will continue to do so. The rate of smoking by females has reached or exceeded that of males and in conjunction with birth control pills and hormone replacement therapy has led to more thromboembolic problems.[13]

There are tools that help in stratifying these patient risks. One of the earliest tools was Rouan's (see below). The Goldman prediction rules have been used by some. Another system has become part of ACLS training over the last decade.

Most of these strategies are based on age, history of previous coronary artery disease, type of pain, EKG findings, cardiac markers, and physical examination findings. Utilizing them will help you determine which patients deserve more complete work ups, monitoring, follow ups, and referrals. It is important to notice that one to four percent of patients with normal EKG findings could have an acute myocardial infarction[14-15] and that cardiac enzymes take from two to six hours to become positive in the majority of cases.[16]

Another tool rating risk assessment came from Braunwald et al in 2000. Its usefulness in the early evaluation of ED patients is somewhat diminished by its complexity and lack of scoring specificity, which seems to target its usefulness to post hoc assessments by consultants rather than the ED physician attempting to stratify the patient.

Rouan's Decision Rule for Myocardial Infarction

Clinical Characteristics (Each count as one point)	
Age greater than 60 years Diaphoresis History of MI or angina Male sex Pain described as pressure Pain radiating to jaw, neck, shoulder, or arms	
Score	Risk of MI (%)
0	Up to 0.6
1	Up to 3.4
2	Up to 4.8
3	Up to 12.0
4	Up to 26.0

Rouan GW, Lee TH, Cook EF, Brand DA, Weisberg MC, Goldman L. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or nonspecific electrocardiograms (a report from the Multicenter Chest Pain Study). Am J Cardiology 1989; 64:1087-92.

Risk Assessment Myocardial Infarction/Unstable Angina/NSTEMI

Short-term high risk for death / myocardial infarction in unstable angina/NSTEMI	Short-term intermediate risk for death / myocardial infarction in unstable angina /NSTEMI	Short-term low risk for death / myocardial infarction in unstable angina/NSTEMI
At least one of:	No high risk features, but must have one of:	No high or intermediate risk features, but may have any of:
History		
Accelerating tempo of ischemic symptoms in preceding 48 hrs	Prior myocardial infarction, peripheral vascular disease, or cerebrovascular disease Coronary artery bypass grafting Prior aspirin use	None indicated
Character of pain		
Prolonged ongoing (>20 min) rest pain	Prolonged (>20 min) rest angina now resolved, with moderate or high likelihood of coronary artery disease Rest angina (<20 min) or relieved with rest or sublingual nitroglycerin	New-onset Canadian Cardiovascular Society class III or IV angina in past 2 weeks without prolonged (>20 min) rest pain, but with moderate or high likelihood of coronary artery disease
Clinical findings		
Pulmonary edema, most likely caused by ischemia New or worsening mitral valve regurgitation murmur S3 or new/worsening rales Hypotension, bradycardia, tachycardia Age over 75 years	Age over 70 years	None indicated
EKG		
Rest angina with transient ST changes greater than 0.05mV Bundle branch block New sustained ventricular tachycardia	T-wave inversions greater than 0.2mV Pathologic Q waves	Normal or unchanged EKG during chest discomfort
Cardiac markers		
Markedly elevated (eg. Troponin T or Troponin I >0.1 ng/mL)	Slightly elevated (eg. Troponin T or Troponin I <0.1 ng/mL)	Normal

From Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST segment elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). J Am Coll Cardiology 2000;36:1062.

TIMI Risk Score for Patients with Unstable Angina and Non-ST Elevation MI: Predictor Variables

Predictor Variable	Point Value of Variable	Definition
Age > 65	1	
> 3 risk factors for CAD	1	Risk factor: • Family history of CAD • Hypertension • Hypercholesterolemia • Diabetes • Current smoker
Aspirin use in last 7 days	1	
Recent, severe symptoms of angina	1	> Anginal events in last 24 hrs
Elevated cardiac markers	1	CK-MB, or Troponin levels
ST deviations > 0.5mm	1	ST depression > 0.5mm is significant ST elevation > 0.5mm for < 20 minutes is treated as ST-segment depression and is high risk ST elevation > 1mm for more than 20 minutes places these patients in the STEMI treatment category
Prior coronary artery stenosis > 50%	1	Risk predictor remains valid even if this information is unknown
Calculated TIMI Risk Score	Risk of > 1 Primary End Point in < 14 Days	Risk Status
0 or 1 2 3 4 5 6 or 7	5% 8% 13% 20% 26% 41%	Low Intermediate High

Taken from the American Heart Association Guidelines CPR/ECC 2005: Handbook of Emergency Cardiovascular Care for Healthcare Providers. Page 38.

The TIMI scoring scheme shown above is currently in widespread use. It must be remembered that, for each patient, the disease is either present or absent and numbers are only a guide based on group studies. It is certain new guidelines will appear ad infinitum and that the trial attorneys will continue to be aware of their existence.

Examination of the Adult Chest Pain Patient

Begin your evaluation of the patient with chest pain by visual observation of the patient and inspection of the vital signs. An emergency department patient should by this time be in a room attached to a cardiac monitor. Do not forget to get O_2 saturation! The ABCs should be attended to, of course; if the patient is stable, then get a 12-lead EKG, put the patient on oxygen if indicated, give an aspirin dose of 325mg (if not allergic), and start the most important part of your assessment, the history.

The history should focus on the onset and evolution of the patient's chest pain. Document its location, quality, severity (using a 1 to 10 scale), radiation, timing, duration, and aggravating and alleviating factors. Pay close attention to any kind of pain that radiates to the jaw, neck, arms, back or abdomen. Associated symptoms such as diaphoresis, weakness, vomiting, nausea, palpitations, and syncope should draw special attention. By this time, and within the ten-minute framework, a 12-lead EKG should be in your hands and, if no contraindications have been discovered (such as hypotension), sublingual nitroglycerine (0.4mg) should be given to the patient with written orders to repeat every two to five minutes times two if chest pain continues and if the systolic blood pressure (SBP) is above 100mmHg. Alternatively a nitroglycerin IV infusion of 10 to 20 micrograms per minute may be used. Titrate to effect, by increasing 5 to 10 micrograms every five to ten minutes until the desired effect is obtained. Nitroglycerine must be discontinued if the blood pressure drops below 90. If pain persists, the use of morphine is indicated; you can use 2 to 4 mg IV (over 1-5 minutes) every 5-30 minutes until the pain improves or the patient develops problems such as hypotension or decreased respiratory rate.[17] Large bore IVs are suggested, as thrombolytics may be required or bolus infusions for hypotension may be necessary. Normal saline is commonly used on a "keep open" basis; if boluses are necessary, 250 to 500mL amounts are commonly given in sequence to bring the pressure back up.

When inspecting the EKG, look for ST elevation, ST depression, Q waves, T wave changes, and bundle branch blocks. Any patient with chest pain and an EKG showing a left bundle branch block (LBBB) needs scrutiny for an acute myocardial infarction. It is very helpful to locate a previous EKG to inspect

for changes, especially in the bundle branch block patients. Solid evidence as to the utility of strategies used to determine the presence of ACS on top of a LBBB remains elusive. Remember that T-wave changes are early signs of ischemia produced by transient hyperkalemia in the ischemic cardiac muscle. Treatment and disposition of the patient is guided by EKG changes and the patient's history, especially their risk factors. Patients (other than those on cocaine) should be given B-blockers (i.e., within the first 24 hours) if no contraindications such as hypotension, bradycardia, heart block (other than first degree), or severe airway disease/pulmonary edema exists.[17] For example, the patient may be given metoprolol 5mg IV in a two to five minute infusion every five minutes times three while in the ED.

The at risk patient population is then divided on the basis of the EKG evaluation into ST elevation MI's (STEMI), non-ST elevation MI's and unstable anginas (NSTEMI/UA), or non-diagnostic EKG patients still with clinical suspicion of ACS. The level of suspicion of cardiac damage and therapy and management is different in each of the three groups. The third group is divided into high risk, intermediate risk, and low risk groups. Patients can move between groups if conditions change, i.e., there is a change in their cardiac markers, their EKG, or their symptomatology.

The management of patients with ST elevations will vary by institution and often depends on access and availability of percutaneous coronary intervention (PCI). There is persuasive evidence that PCI is more effective than fibromyalgia if it can be accomplished within the 90 minutes time frame.[18] In many parts of rural America, patients with ST elevations will benefit from thrombolytic therapy if PCI is not available within 90 minutes (after making certain to document the presence or absence of contraindications). Remember every minute counts as "time is muscle." The national goal for administration of fibrinolysis is 30 minutes and door to PCI is 90 minutes.[19] Make sure to document any factors that prevent you from achieving these goals. If you are not fortunate enough to be at a cardiac center, then arrange for appropriate consultation or transfer expeditiously. Anti-coagulant, anti-platelet, and fibrinolytic therapy should be considered and discussed with the receiving physician. If appropriate, knowledge of the receiving physician's preferences and standing protocols are quite helpful, although by now the standard of practice allows emergency physicians to deliver such care using widely distributed protocols without direct supervision or specific recommendations by cardiologists.

Most clinicians order the following laboratory and ancillary tests in order to more fully evaluate patients who often have multiple medical problems, are on a myriad of medications, and are unknown to the emergency physician. Such test include: complete blood count, chemistry profile, liver panel, troponin, myoglobin, CK-MB markers, TSH and coagulation studies, though there is abundant evidence that results from these tests rarely change the immediate therapy or management.

Serial EKGs and cardiac markers are standard over a period of time (8-12 hours) although once positive the patient can be stratified and referred. Some institutions use continuous EKG monitoring, though the value is uncertain. Otherwise, the EKGs should be repeated at any time if symptoms warrant, or at least every two to four hours.[2] Of the cardiac markers, myoglobin rise is the earliest to be detected and is very sensitive though not very specific; frequently increases are due to non-specific factors such as renal impairment and muscle contusions. Myoglobin levels may be useful for ruling out acute myocardial infarction if the levels remain normal during the first several hours (4 to 8 hours). Troponins and CK-MB may take more than four hours to rise and may stay positive for quite some time; in the case of troponin up to two weeks. The risk of death due to a MI correlates with the quantitative value of the troponin.[16] After excluding an acute coronary syndrome (i.e., STEMI, NSTEMI/UA) the workup for non-acute coronary syndrome and non-cardiac chest pain can be started. These workups can be accomplished by referral for provocative testing and may take place on an outpatient basis.

Alex Agostini-Miranda, M.D., completed the Family Practice Residency Program at the University of Puerto Rico. He currently has privileges at the Miami VA Hospital and at Kendall Regional Medical Center (ER practice). Dr. Agostini-Miranda also holds an academic appointment of Auxiliary Professor at the University of Puerto Rico.

Loren Crown, M.D., is a clinical professor at the University of Tennessee. Currently, he is the medical advisor for the graduate training programs in emergency medicine in Jackson and Memphis.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist.

References

1. Cannon CP. Acute Coronary Syndromes: Risk Stratification and Initial Management: Cardiol Clin 2005;23:401-409.
2. Pope JH, Selker HP. Acute Coronary Syndrome in the Emergency Department: Diagnostic Characteristics, Test, and Challenges: Cardiol Clin 2005;23:432-51.
3. American Heart Association. Heart and stroke statistics-2005 update. Dallas, TX: American Heart Association; 2004.
4. Eslick GD, Fass R. Noncardiac chest pain: evaluation and treatment: Gastroenterol Clin N Am 2003;32:531-52.
5. Eslick GD, Talley NJ. Non-cardiac chest pain: squeezing the life out of the Australian healthcare system? Med J Aust 2000;173:233-4.
6. Cayley WE. Diagnosing the Cause of Chest Pain: Am Fam Phisycian 2005;72:2012-21.
7. Cava JR, Sayger PL. Chest pain in children and adolescents: Pedriatr Clin N Am 2004;51:1553-68.
8. Towbin JA. Molecular genetics of sudden cardiac death. Pediatr Clin N Am 2004;51:1229-55.
9. Selbst SM. Evaluation of Chest Pain in Children: Pediatr Rev 1986;8(2):56-62.
10. Eslick GD, Coulshed DS, Talley NJ. The burden of illness of non-cardiac

chest pain. Aliment Pharmacol Ther 2002;16(9):1677-82.

11. Kahn S. The challenge of evaluating the patient with chest pain. Arch Pathol Lab Med 2000;124:1418-9.
12. Limkakeng, AT. Chest Pain: Ask the Right Questions to Get Useful Information. ACEP News, June 2006.
13. Reichert VC, Seltzer V, Efferen LS, Kohn N. Women and tobacco dependence. Med Clin N Am 2004;88:1467-81.
14. Boie ET. Initial Evaluation of Chest Pain: Emerg Med Clin N Am 2005;23:937-57.
15. Brady WJ, Roberts D, Morris F. The nondiagnostic ECG in the chest pain patient: normal and nonspecific initial ECG presentations of acute MI. Am J Emerg Med 1999;17(4):394-7.
16. Achar SA, Kundu S, Norcross WA. Diagnosis of Acute Coronary Syndrome: Am Fam Physician 2005;72:119-26.
17. Field JM, Hazinski MF, Gilmore D. Handbook of Emergency Cardioascular Care for Healthcare Providers from the American Heart Association. 2006.
18. Jancin, B. Nearly 80% of Adults Live Within One Hour of a PCI Hospital. ACEP News, June 2006.
19. Pollack CV, Roe MT, Peterson ED. 2002 update to the ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: Implications for emergency department practice: Annals of Emergency Medicine 2003;41(3).

Sidebar

1. While not all cases of chest pain turn out to be heart attacks, when you are wearing a white coat, treat all chest pain as such during the early management phase.
2. In chest pain, time is muscle, but also, time is money. You are being timed! If you fail to diagnose and treat according to CMS and other standards in the future, funding may be withheld to the institution.
3. As soon as the chief complaint of chest pain is documented, GET INVOLVED! You, as the captain of the ship, will be accountable from the moment that the patient gets into your building. Make sure the staff understands this.
4. Teach and train all health-related personnel in your area to recognize the signs and symptoms of a heart attack. The nurse, respiratory technician, janitor, and receptionist are people who may interface with patients at any time, and when they tell you something, heed their advice. Remember, you may be the new doctor, but the people in your work area will have had many years of experience.
5. When you see a patient who declares chest pain, look, listen, and feel. If you think that the patient is not stable, give treatment immediately; put them on a stretcher, and let them "lie with MONA" (M=morphine, O=oxygen, N=nitroglycerin, A=aspirin).
6. Patients with high-risk scores who have histories of diabetes, hypertension, lipid disorders, and thrombotic events must be evaluated very thoroughly. This means appropriate observation, follow up, including stress tests, 2D-Echos, nuclear medicine scans, consultations, and so forth.
7. Once you start the work up for chest pain, finish it. One test or set of markers is never enough. Medico-legally you will find few experts to testify in your favor if you "rule out" an acute coronary syndrome with a single cardiac marker. If the patient tries to leave, make them sign out AMA and clearly document the steps taken.
8. Know your limits. If you are in a facility (non PCI, non CABG) that cannot manage the problem the patient has, TRANSFER the patient. Do not ignore EMTALA. Many experts urge PCI if access can be accomplished rapidly; most everyone recommends lytics, anti-platelet drugs, and anti-coagulants early. Know your local referral options.
9. Get prepared. Make sure you know what is available in your hospital and where the equipment and medications in your department are. Check the intubation equipment, i.e., light bulbs, and make sure you have the proper blade tube sizes and an assortment of back-up airway devices.
10. Wall-mount the ACS protocols so that you can find them rapidly, check them frequently, and keep your information current. While ACLS may have detractors, it is a good place to start.

sounding board

Emergency Medicine Afield: The Russian Federation

James Meade, M.D., FAAEP

I recently traveled in the Russian Federation on a tour with my college alumni. I visited the emergency department of the largest hospital in St. Petersburg, the Elizavetinsky, and met with the director of the hospital. There is no formal training in emergency medicine or any certification of emergency physicians in the Russian federation. Their physical plants suffer from severe lack of funds for equipment and salaries. As a result, the health of the population has deteriorated, with falling population, birth rate, and longevity.

On a previous visit with the International Cardiovascular Society, 10th International Congress in 1971, I visited several hospitals in Moscow. That was the first International Medical Meeting in the Soviet Union, headed by Dr. Denton Cooley of the Texas Heart Institute, and we saw what was the best of the best, with specialized hospitals for cardiovascular and orthopedics. They were clean, well maintained, with intelligent and energetic staffs (who were chain smokers, even on ward rounds).

In spite of that, I found the atmosphere so depressing that I cut my tour short and left early. On my last day, a new friend, a cardiologist, took me for a ride to the Lenin Hills overlooking the city of Moscow and said, "Of course, you must come back soon." I did not have the heart to tell him I would never come back to the Soviet Union.

However, times have changed over the past 36 years and the Soviet Union is gone, replaced by the Russian Federation. I recall walking across Red Square at midnight (at that time Moscow was the safest city in the world, as long as your papers were in order) and watching the changing of the guard at Lenin's tomb. I saw young blond men doing the "goose-step" as they were coming out of the east gate of the Kremlin and changing places with the guards at the door of the Mausoleum. Overhead, flood lit, waved the hammer and sickle flag. If someone had told me then that this would all self-destruct in 20 years, I would have said they were mad, this empire will stand for another hundred years.

Now, the red, white, and blue flag of the Russian Federation flies over the Kremlin, and the country is split between the mass of people, who are poverty stricken, and a few plutocrats, who drive Porsche SUVs. The Lenin Hills are now the Sparrow Hills. Beggars, hucksters, and pickpockets are everywhere, something never seen in the Soviet Union. The average wage of a physician is comparable to US $200-250 per month, which is not enough to live on. Here in the United States young people live with their parents by choice, in the Russian Federation by necessity. For instance, our guide was a 40-year old woman, who was a teacher, but working as a tour guide in the summer. Her husband was a major in the army. There were five wage earners in her extended family, one of which was a doctor. With their combined incomes, they were able to survive.

Since the 1970s medical care has severely deteriorated in the Russian Federation, in spite of the fact that the constitution guarantees free health care for all citizens, and Russia has more physicians and health care workers than any other country in the world. The population is dropping rapidly from a peak of 148 million to 142.3 million in 2006, due to falling birth rate. Male longevity is down to an average of 60 years. Actually, this has slightly improved from 55 years in 1994. This is mainly due to complications of alcoholism and violence. The Russians smoke as much as ever. The problem of heavy alcohol consumption has been recognized since 1998, but little has been accomplished to rectify the situation. President Gorbachev attempted to limit alcohol consumption, but this resulted in illegal consumption of anything with any kind of alcohol, with devastating health results. Today, there is no problem getting vodka, the most common Russian drink. Even drinks such as Absinth, which has been illegal in the United States[1,2], are available.

The most recent UN report states that Russia is "doing too little to reverse a critical decline in its population" and predicts that the population "could fall to 100,000,000."[3]

For comparison, the United States population is 303,000,000, with population growth at 1% and a birth rate of 2.1, while the Russian federation has a population of 142,000,000, with a -0.5% growth and a birth rate of 1.4. There are 15 deaths

	UNITED STATES	RUSSIAN FEDERATION
POPULATION	303,000,000	142,300,000
GROWTH	1%	-.05
DEATH RATE	8.3/1000	15/1000
INFANT MORTALITY	6.4/1000 births	15.1/1000 births
FERTILITY RATE	2.1	1.4
LONGEVITY MALE	77.1 Years	60.0 Years

Table 1. Decline in Russia's Population

per 1000 people, and infant mortality is 15.13/1000. Needless to say, the Russians have no problem with illegal immigration, since no one wants to move there (See table 1)[4].

The birth rate is falling and is now 1.4, which is not enough to sustain population growth. The death rate is 15/1000, compared with 8.3/1000 in the United States.[4] There are more abortions than live births, 1.6 million to 1.5 million. Former President Vladimir Putin has called the declining population "the most acute problem of contemporary Russia."[2] The governor of the province of Ulyanovski gave everyone a day off in February for "Family Contact" to encourage more pregnancies.

Even worse is the problem of HIV/AIDS, which is the highest in the Western world in Russia and the Ukraine. This is primarily due to intravenous drug use, but heterosexual spread is increasing. Due to poverty, young women are forced into the sex trade, which masquerades as "dating services" or "marriage agencies" but are really brothels without walls run by criminals.

The office of the Consulate General for the United States in St. Petersburg currently warns that:

"Western medical care in St. Petersburg can be expensive and difficult to obtain. . . Some facilities offer quality services but many restrict services to normal business hours and to persons willing to pay for services in advance."

"The Russian national medical system provided emergency care that, while officially free of charge, may be of a quality that ranges from uncomfortable to unacceptable. Moreover, Russian doctors may demand payment for disposable needles, medications, and other services."

However, this is denied by those physicians that I talked to.

The Elizavetinsky Hospital is located in St. Petersburg and is a 1025 bed hospital (which can house up to 1250 patients in the winter) and covers an area of 1 million people; supposedly they have 200 patients a day in the ED, but on the day that I was there, it seemed deserted.

I took a taxi from my hotel, which was about an hour away. It is block after block of decrepit Soviet-era apartment houses. We pulled up to a weed-grown lot before a small decrepit building. "Is this it?" I asked, being reluctant to get out of the cab and be stranded. The driver pointed to the sign, which indeed said Elizavetinsky Hospital (fortunately I can read Russian). It was the gatehouse, and I showed the caretaker my note and got a curt nod and a motion to go around the corner.

Eventually, I was able to find the office of the chief physician, Dr. Boris Tuites, with the aid of his interpreter, Dr. Anastasia Timoshenko. We discussed the education of physicians in the Russian Federation and that of emergency physicians in particular.

Medical education in the Russian Federation is six years after secondary school, followed by one year of what is an essentially rotating internship. Specialty training is about two years, which for them is a practice track. There is no emergency medicine residency or certifying examinations. All education is free with admission by examination.

I have always been impressed by the dedication and expertise of the physicians that I have met in Russia, but unfortunately they are hampered by their lack of money and equipment. Stretchers are old and rusty. Sheets and blankets are in short supply.

Figure 1: Rusty stretchers in the waiting area

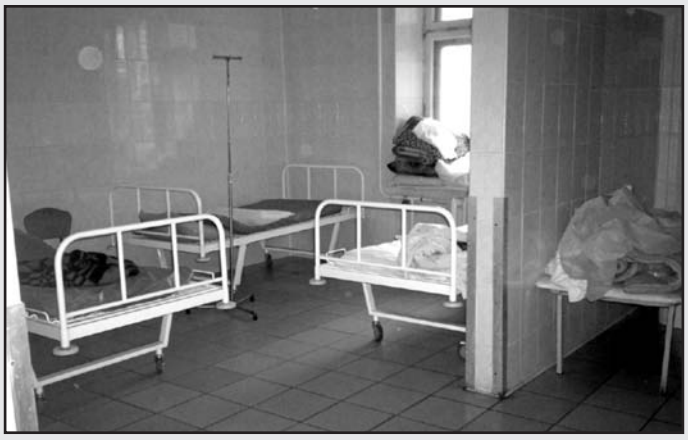


Figure 2: Sheets and blankets are in short supply

Figure 3: Plan of the emergency department

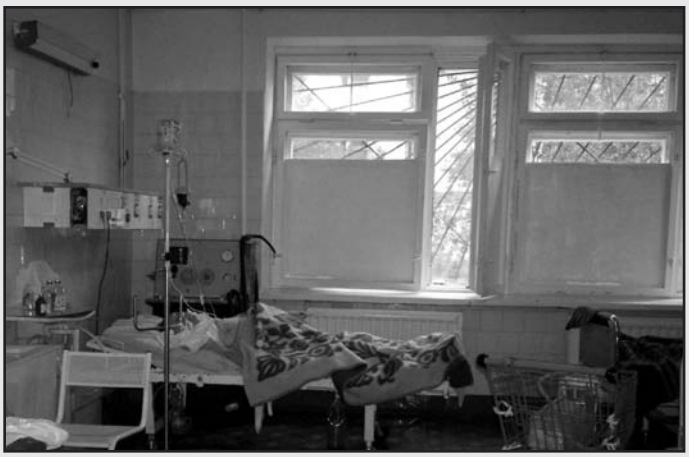


Figure 4: The ICU

I was taken on a tour of the Emergency Department, which takes up the entire lower floor of the hospital, most of which seemed deserted. There are supposedly over 300 physicians and over 600 nurses who are employed by the hospital.

The emergency physician essentially is a triage officer, who takes care of minor problems and calls consultants when they are needed for more complex cases. Unlike in the United States, there is no trouble getting consultants, since they are all in-house and available around the clock. All of the major specialties have their own rooms dedicated to their needs.

The ambulance service is fine in theory. The Elizavintsky has three ambulances parked in the bay with no active calls. They usually have a driver and what corresponds to a paramedic and a physician. Only about 30% of calls are transported to the hospital, the rest taken care of on the spot. Once in the hospital emergency department they are seen by a nurse for triage and then evaluation by the emergency physician, who will decide which specialist to call. Since the specialists are all present in the hospital, there is a minimal delay in getting services. The director stated that the goal is to have all patients seen and treated within two hours.

The excellent specialty hospitals that I saw on my last visit were geared to the treatment of communist VIPs, who suddenly disappeared along with the large amounts of money needed to run these establishments. I was totally unable to get in touch with or get a visit to any of the hospitals that I had toured before.

In spite of the dedicated and intelligent physicians that I met, my impression is that, if you become seriously ill in the Russian Federation, you should seek transfer elsewhere. In St. Petersburg this usually means the University Hospital of Helsinki or the American Hospital in Paris. In the past, patients have been able to go the Department of Defense hospital in Frankfurt, but apparently this is no longer possible due to the pressures of the Iraq war. If you have travel insurance, it is important to read the fine print, since most travel policies only cover transport to the nearest medical center capable of taking care of your problem. This may mean going from bad to slightly less bad. What you want is repatriation to the United States, which very few policies provide for. One is the AMA program (only for AMA members who carry AMA insurance), which will repatriate after stabilization. No one will transfer an unstable patient. Another is AIG Travel Guard provided that you have purchased insurance for a single trip.

On my last day in Moscow there was a celebration by the armed forces paratroopers. The streets were filled with drunken young men, looking for trouble. It reminded me of Germany in the 1920s, a once great nation fallen on bad times, with a large group of angry young men looking for an outlet for their frustrations in violence. It only requires a charismatic leader to unite them. The Russian Federation is a nuclear powder keg waiting for the opportunity to explode.

Summary

Medical care in the Russian Federation has markedly deteriorated since the fall of the Soviet Union with decreased longevity (among males, due to trauma, alcoholism and suicide), falling birth rate and population, decrepit facilities, and shortages of supplies and drugs. The AIDS epidemic is the worst in the western world. Emergency medicine is not a recognized specialty, and there is no specialized training or certification in the field.

James Meade, M.D., Board Certified in Emergency Medicine, practiced in Syracuse, NY, where he was Clinical Assistant Professor of Surgery at Upstate Medical Center. Dr. Meade, retired from clinical practice, currently resides in Sarasota, FL. He continues to be active in the oral examination process and in the political realm in Tallahassee.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The author has stated that no such relationships exist.

References

1. Rosenberg M. Population Decline in Russia, http://www.about.com: geography May 31, 2006. Vlassof, Vasiliy: The Role of alcohol and social stress in Russia mortality rate. JAMA 1999; 281;321.
2. Leon DA, Shkolnikov VM. Social stress and the Russian mortality crisis. JAMA 1998; 279; 790-791.
3. Reuters:Russia: U.N. Warning on Population. The New York Times April 29, 2008 Section A, page 6.
4. RAND/Monogram reports/DaVanzo, J and Grammich, C:Dire Demographics RAND Santa Monica CA page 13 Summary Consulted 7/5/2008.
5. http://www.about.com: GEOGRAPHY UNITED STATES CENSUS AND DEMOGRAPHICS consulted 7/5/2008.
6. Background Note: Russia. Bureau of European and Eurasian Affairs. US Department of State February 2007 http://www.state.gov/r/pa/ei/bgn/3138.htm.

Manuscript Criteria and Information

The American Journal of Clinical Medicine (AJCM), the official journal of the American Association of Physician Specialists, Inc. (AAPS), is a peer reviewed journal dedicated to improving the clinical practice of medicine by publishing educational and informational articles. The AJCM is the official journal of the American Association of Physician Specialists, Inc. (AAPS).

Send all manuscripts via email to editor@aapsus.org.

Manuscripts received are not to be under simultaneous consideration by another publication. Accepted manuscripts become the permanent property of the American Journal of Clinical Medicine and may not be published elsewhere without permission from the publisher. Manuscripts submitted by mail to the Journal will NOT BE RETURNED.

Authorship Responsibility, Financial Disclosure, Assignment of Copyright, and Acknowledgment Forms: Authorship responsibility forms must be completed and signed by each author and accompany submitted manuscripts. Each author must submit a statement that specifies whether he or she has financial or proprietary interest in the subject matter or materials discussed in the manuscript. These forms may be downloaded from the AAPS website www.aapsus.org or may be obtained by request to the AAPS office at 813-433-2277 ext 30.

Authorship Responsibility: All accepted manuscripts are copyedited and an edited typescript is sent for the author's approval. The author is responsible for all statements in the work, including the copy editor's changes.

Data Access and Responsibility: For reports containing original data, at least one author (e.g., the principal investigator) should indicate that he or she "had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis" (DeAngelis CD, Fontanarosa PB, Flanagin A. Reporting financial conflicts of interest and relationships between investigators and research sponsors. JAMA. 2001;286:89-91).

Units of Measure: Conventional units of measure are preferred, with Système International (SI) units expressed secondarily (in parentheses). In tables and figures, a conversion factor to SI may be presented in the footnote or legend to economize space. Exceptions to this policy include calories, hematocrit, glycosylated hemoglobin, blood cell counts, and ejection fraction, for which conventional units alone should be expressed. The metric system is preferred for length, area, mass, and volume.

Manuscript Preparation: Manuscript preparation should generally follow the guidelines outlined in The International Committee of Medical Journal Editors: "Uniform requirements for manuscripts submitted to biomedical journals", The Journal of the American Medical Association, March 19, 1997;277:927-934. An abstract of 100-150 words is required. The main text should be narrative in form and should be broken up into appropriate headings and/or subheadings. Any abbreviations used should be completely defined upon the first usage. The style of writing should conform to acceptable English usage and syntax. Please avoid slang, medical jargon, obscure abbreviations, and abbreviated phrasing.

Manuscripts should be submitted electronically online to the email address above as a Microsoft Word document. Authors' names should be on the title page ONLY. Revisions, Editorials, and Editorial Correspondence follow the same procedures outlined, including a word count.

Title Page: All submissions must include a title page. Titles should be concise, specific, and informative, and should contain the key points of the work. Authors' names should be on the title page only. Include the full names, degrees, and academic affiliations of all authors, indication of the corresponding author, his or her address, phone, fax, and e-mail address, the address for reprint requests, and, if the abstract or any portion of the manuscript was presented at a meeting, the name of the organization, place, and date on which it was read. Include a word count for text only, exclusive of title, abstract, references, figure legends, and tables. Include brief biographical information including current position. Financial disclosure information should be included as a footnote.

Acknowledgment Section: List all persons who have made substantial contributions to the work reported in the manuscript (including writing and editing assistance), but who are not authors; any financial interest in the subject matter or materials discussed in the manuscript; any research or project support/funding; any grant support. Manuscripts with statistical evaluations should include the name and affiliation of statistical reviewer(s).

Original Research: For authors who wish to submit original research, including reports of randomized controlled trials, please contact the editor-in-chief for instructions and criteria for publication.

References: List references numerically (not alphabetically). All subsequent reference citations should be to the original number. Cite all references in the text or tables. Unpublished data and personal communications should not be listed as references. References to journal articles should include (1) author(s) (list all authors and/or editors up to three; if more than three, list first three and "et al"), (2) title, (3) journal name (as abbreviated in Index Medicus), (4) year, (5) volume number, and (6) inclusive page numbers. References to books should include (1) author(s) (list all authors and/or editors up to six; if more than six, list first three and "et al"), (2) chapter title (if any), (3) editor (if any), (4) title of book, (5) city of publication, (6) publisher, and (7) year. Volume and edition num-

bers, specific pages, and name of translator should be included when appropriate. The reference numbers in the reference list (if any) should be keystroked. Do not let the word processing program generate the reference numbers, using such features as automatic footnotes or endnotes. The author is responsible for the accuracy and completeness of the references and for their correct text citation. Please notice how reference is set in text in example below. Set yours to match.

Reference in Text: The following is an example of how to list references within the text: "Aeromedical evacuation operations, conducted with either helicopters or fixed-wing aircraft, operate in various environmental conditions, making these operations inherently dangerous and hazardous."[21-23]

Do not include "personal communications" in the list of references. Authors who name an individual as a source for information in a personal communication, be it through conversation, a letter, e-mail message, or telephone call, should obtain written permission from the named individual.

Format: Articles should be submitted in Times New Roman 10pt. font, single spaced with no additional or unnecessary styles applied to text.

Tables, Illustrations, Legends: Number all tables and illustrations in the order of their citation in the text. Include a title for each table and figure – a brief, succinct phrase, preferably no longer than 10 to 15 words. Keep in mind all tables, illustrations and legends will be printed in grayscale and color coded images may be difficult to interpret.

Tables: Title all tables and number them in order of their citation in the text. Double-space each table on separate sheets of standard size white paper. If a table must be continued, repeat the title on a second sheet, followed by "cont."

Illustrations: Illustrations should be submitted online as a separate document. Most standard programs will be accepted. Please refer to the next section for details.

Digital Art Submissions: Digital art must be submitted electronically online as a separate file from the manuscript. Calibrated color proofs should be submitted with color digital files, if possible. The canvas size of continuous-tone images should be at least five inches wide (depth not important) with an image resolution of at least 300 dpi. Line art images should have a minimum resolution of 1270 ppi. Formats accepted are EPS, TIFF, and JPG. Keep in mind all tables, illustrations, and legends will be printed in grayscale and color-coded images may be difficult to interpret.

Legends: Include double-spaced legends (maximum length 40 words) on separate pages. Indicate magnification and stain used for photomicrographs and method of enhancement for digitally enhanced images.

Photographic Consent: A letter of consent must accompany all photographs of patients in which a possibility of identification exists. It is not sufficient to cover the eyes to mask identity.

Acknowledgments: Acknowledge illustrations from other publications and, when applicable, include author(s), title of article, title of journal or book, volume number, page(s), month, and year. The publisher's permission to reproduce in print and online and in AJCM licensed versions should be submitted to the AJCM when the manuscript is submitted.

Disclaimer: Publication of any article or statement in the AJCM does not constitute an endorsement by the AJCM or its editors. Publication of any advertisement in the AJCM does not constitute an endorsement by the AJCM or its editors.

Manuscript Submission Checklist

❑ Submit manuscript electronically online as a Microsoft Word document to editor@aapsus.org. Leave right margins unjustified (ragged).

❑ On the title page, designate a corresponding author and provide a complete address, telephone, fax numbers and e-mail address. Authors' names should be on the title page ONLY. This allows reviews to be anonymous. Each author must also include current employment/position information, and any other biographical information, which author wishes to be included at the end of the article.

❑ On the title page, include a word count for text only, exclusive of title, abstract, references, tables, and figure legends.

❑ Complete Authorship Responsibility Form, which includes Financial Disclosure, Assignment of Copyright and Acknowledgement.

❑ Include statement signed by corresponding author that written permission has been obtained from all persons named in the acknowledgment (if applicable).

❑ Include research or project support/funding in an acknowledgment (if applicable).

❑ Check all references for accuracy and completeness. Put references in proper format in numerical order, making sure each is cited in sequence in the text. Please see In-Text Example above and make sure your references are set the same way.

❑ Include a title for each table and figure – a brief, succinct phrase, preferably no longer than 10 to 15 words.

❑ Submit illustrations electronically online in a file separate from the manuscript.

❑ For digitally enhanced images, indicate method of enhancement in legend and submit electronically online.

❑ Include informed consent forms for identifiable patient descriptions, photographs, and pedigrees (if applicable).

❑ Include written permission from publishers (or other copyright owner) to reproduce or adapt previously published illustrations and tables (if applicable).

NEW THIS YEAR

We Really Want You To Attend the 2009 Annual Scientific Meeting so...

We've listened to your comments and made some changes. We've made it more...

Meaningful Organized Effective Informative Fun!

BOARD OF DIRECTORS MEETING----------

We strongly encourage everyone to attend this meeting on June 25, 2009. The Board of Directors will convene prior to the Opening Session. You will hear the latest Board decisions and gain valuable information about AAPS policies, directions, and future developments. .

OPENING SESSION ----------

Then join us for a General Membership Luncheon, followed by the **Opening Session** in the afternoon.

We have added this important new component – the **Opening Session** – so that you can learn about Board decisions and eliminate repetition in other meetings.

ORIENTATION SESSION----------

There will be an **Orientation Session** for newly elected/appointed Academy and Board members.

This required session for all newly elected/appointed members of Academies and Boards will outline the responsibilities, expectations, and procedures of the Association.

CMEs ----------

We'll provide the complete schedule of **CME** offerings so you can plan your time more effectively. By participating in activities that offer **CMEs**, you may earn the following:

Up to 16.0 CMEs Scientific Presentation sessions
Up to 14.0 CMEs All eligible Certification Workshops
Up to 7.5 CMEs Medical Ethics course

REGISTRATION----------

We've provided you with more registration choices – the sooner you register, the more you will save.

Take advantage of **EARLY** registration. Just complete the registration form and send with full payment by April 30, 2009.

FLASH DRIVES ----------

Convenience: Instead of a bulky paper version of the scientific presentation materials, we'll provide a flash drive for your computer.

LOEWS CORONADO BAY RESORT AND SAN DIEGO ----------

FUN! Take advantage of the sights and attractions offered in Coronado and San Diego. See the famous Hotel Del Coronado. Visit the San Diego Zoo, Gaslight Quarter for restaurants and shopping, visit the USS Midway, take a gondola ride. There's something for everyone!

REGISTRATION FORM: AAPS 2009 House of Delegates and Annual Scientific Meeting

TO REGISTER: FAX: 813-830-6599 (ONLY if paying by Credit Card) • MAIL: AAPS, 5550 W. Executive Drive, Suite 400, Tampa, FL 33609-1035

Diplomate in: _____ ID# (if available) _____

Name: _____ Address: _____

City: _____ State: _____ Zip: _____

Cell Phone: _____ Work Phone: _____ Work Fax: _____

Home Phone: _____ Home Fax: _____ Email Address: _____

Special Needs (Diet, Accessibility): _____ For (Name of Person): _____

DIPLOMATES ONLY: WRITTEN AND ORAL EXAMINATION DEVELOPMENT WORKSHOP REGISTRATION: Please check the session(s) you plan to attend.

DIPLOMATES WHO PLAN TO PARTICIPATE IN CERTIFICATION WORKSHOPS MUST REGISTER IN ADVANCE FOR ALL CERTIFICATION WORKSHOPS. NO EXCEPTIONS WILL BE MADE.

❑ JUNE 25 **ITEM WRITING WORKSHOPS FOR ORAL EXAMINATIONS** Specialty Name: _____

❑ JUNE 25 **ONLINE AUTHORING WORKSHOP** Specialty Name: _____

❑ JUNE 27 **EXAMINATION DEVELOPMENT WORKSHOPS FOR ORAL EXAMINATIONS** Specialty Name: _____

❑ JUNE 27 **EXAMINATION DEVELOPMENT WORKSHOPS FOR WRITTEN EXAMINATIONS** Specialty Name: _____

(Open to those recertifying in years **other than** 2009 and 2010)

BY INVITATION ONLY

❑ JUNE 26 **WRITTEN EXAMINATION DEVELOPMENT WORKSHOP FOR EMERGENCY MEDICINE** (Open to those recertifying in years **other than** 2009 and 2010)

❑ JUNE 27 **ORAL EXAMINATION DEVELOPMENT WORKSHOP FOR EMERGENCY MEDICINE**

MEDICAL ETHICS COURSE REGISTRATION: (SEPARATE REGISTRATION FEE APPLIES)

Per Person $450 x _____ (# attending) = $ _____

MEDICAL ETHICS REGISTRATION SUBTOTAL _____

PHYSICIAN REGISTRATION: (Name of Physician) _____ (Includes CMEs for scientific sessions and workshops attended, conference materials, breakfasts, luncheons, Welcome Reception, and President's Reception & Awards Dinner.)

EARLY REGISTRATION • ALL FEES RECEIVED IN FULL PRIOR TO APRIL 30, 2009

❑ AAPS Member $1190 x ___ (# attending) = $_____

❑ AAPS Member Speaker $890 x ____ (# attending) = $_____

❑ AAPS Retired/Life Member $790 x ____ (# attending) = $_____

❑ Non Member CME Only $1190 x ___ (# attending) = $_____

STILL-TIME-TO-REGISTER • ALL FEES RECEIVED IN FULL PRIOR TO JUNE 12, 2009

❑ AAPS Member $1390 x ___ (# attending) = $_____

❑ AAPS Member Speaker $1090 x ___ (# attending) = $_____

❑ AAPS Retired/Life Member $990 x ____ (# attending) = $_____

❑ Non Member CME Only $1390 x ___ (# attending) = $_____

LATE/ON-SITE REGISTRATION • ALL REGISTRATIONS AFTER JUNE 12, 2009 ***ONLY CREDIT CARD PAYMENTS ACCEPTED***

❑ Late/On-Site Registration $1780 x ___ (# attending) = $_____

PHYSICIAN REGISTRATION SUBTOTAL _____

SPOUSE/GUEST/CHILDREN REGISTRATION (Includes breakfasts, Welcome Reception, President's Reception & Awards Dinner, and official name badge. **Lunches are not included.**)

SPOUSE/GUEST/CHILDREN OVER AGE 12 ..... $425 x _____ (# attending) = _____

Name of Spouse/Guest/Child: _____ Name of Spouse/Guest/Child: _____

CHILDREN UNDER AGE 12 REGISTRATION (Includes breakfasts, Welcome Reception, and official name badge.)

CHILDREN UNDER 12 ..... $0 x _____ (# attending) = $0

Name of Child: _____ Age: _____ Name of Child: _____ Age: _____

SPOUSE/GUEST/CHILDREN REGISTRATION SUBTOTAL _____

ADDITIONAL TICKETS

WELCOME RECEPTION ..... $125 x ____ (# attending) = _____

Name of Spouse/Guest/Child: _____ Name of Spouse/Guest/Child: _____

PRESIDENT'S AWARDS DINNER ..... $125 x ____ (# attending) = _____

Name of Spouse/Guest/Child: _____ Name of Spouse/Guest/Child: _____

ADDITIONAL TICKETS SUBTOTAL _____

GRAND TOTAL _____

All registrations after June 12, 2009, including on-site, must be paid in full prior to admission to any AAPS activities. *Due to the preparations required prior to the Annual Meeting and the guarantees that AAPS must provide to the hotel in advance, AAPS strongly encourages you to register for the meeting and reserve your hotel rooms early.

MEETING REGISTRATION PAYMENT

❑ Check (enclosed) ❑ MasterCard ❑ Visa ❑ American Express **Total to be charged**: _____

Cardholder's Name: _____ Card Number: _____ Expiration Date: _____

Cardholder's Mailing Address: (if different from address on previous page) _____

Cardholder's Signature: _____

Hypercalcemic Crisis: A Case Study

Loren A. Crown, M.D.
Andra Kofahl, EMT-P
Robert B. Smith, M.D.

Abstract

A 46-year-old female in an acute delusional state arrived by EMS to the Emergency Department. She was hypertensive, hyponatremic, hypokalemic, and most importantly, hypercalcemic with an extremely elevated lipase level. The causes and treatment of hypercalcemia are reviewed in this case study.

Introduction

Regulation of serum calcium, within a range of 8.5 to 10.5 mg/dl, is tightly managed by two hormones, parathyroid hormone (PTH) and calcitriol. Several factors can affect the body's ability to maintain homeostasis. More than ninety percent of hypercalcemia is caused by primary hyperparathyroidism (HPT) or malignancy. Determining the etiology can be daunting during a hypercalcemic crisis due to the difficulty of obtaining an accurate history in the presence of the neurological dysfunction common when calcium levels are greater than 12 mg/dl. Symptoms may also include muscle weakness, constipation, anorexia, nausea, vomiting, and abdominal pain. Other concurrent electrolyte abnormalities may coexist. Less common findings, possibly seen in a hypercalcemic crisis when levels reach 15 mg/dl or more, are pancreatitis, peptic ulcers, hypertension, and cardiac effects (bradycardia or shortened QT intervals). If left untreated, hypercalcemic crisis can eventually lead to renal failure and/or coma.

Treatment consists of rapid correction of hypercalcemia through intravenous hydration and loop diuretics, provided renal function is adequate. Several medications can also help maintain calcium levels. However, if the patient is in renal failure, dialysis must be considered. Ultimately the cause must be identified and treated.

Presentation of Case

A 46-year-old woman, stopped by airport police for erratic driving, was sent by ambulance to the Emergency Department (ED) with an initial complaint of generalized confusion which she stated started approximately 24 hours prior. She was at the airport to meet her daughter, who was due to arrive at 1300, but the actual time she was detained by the officers was 2030 (7 ½ hours after the pickup time). The daughter was contacted by phone and was able to provide a past medical history of alcohol abuse, hepatitis B, and hypertension. The patient was unable to recall her prescribed medications; she denied recent illicit drug or alcohol use or any allergies to medications. Her appearance was that of a clean, well-groomed individual. She was extremely lethargic and fell asleep during her examination. Her initial vital signs were: Glasgow Coma Score (GCS) 15, blood pressure 199/118 mmHg, heart rate 77 beats per minute, respiratory rate of 18 per minute, an oxygen saturation of 98% on room air, and a temperature of 97.8 F orally. Cardiac monitoring revealed sinus rhythm in the 70s without ectopy.

The patient's physical exam was essentially unremarkable with no focal motor or sensory deficits. Laboratory studies included the following: comprehensive metabolic panel, complete blood count, cardiac enzymes, serum drug screen, and urinalysis. Bedside glucose reading was 115 mg/dl. No odors of acetone or alcohol were present and a serum alcohol was negative. In addition, an electrocardiograph (EKG), chest x-ray, and computed tomographic (CT) scan of the head were ordered. Results showed electrolyte derangements as seen in Table 1. The EKG interpretation was normal sinus rhythm with nonspecific repolarization abnormality; the CT and chest x-ray were negative.

Serum Drug Screen Results		
Acetaminophen (ug/ml):	**<10.0**	**10.0-20.0 Ref.**
Alcohol, Ethyl (mg/dl):	**<10.0**	**0.0-10.0 Ref.**
Salicylate (mg/dl):	**<1.0**	**2.0-29.0 Ref.**

Urine Drug Screen Results		
Urine Analyze	**Negative**	**Negative Ref.**

Hormone Results		
Cortisol (ug/dl)	**18.73**	**3.09- 22.40 Ref.**
PTH, intact (pg/ml)	**12.0**	**14-72 Ref.**
TSH (uIU/ml)	**2.290**	**0.35-5.50 Ref.**

Table 1: Laboratory Trends

Complete Blood Count

Results: Units: Low Ref: High Ref:	WBC 1000/mm3 4.0 11.0	RBC 10x6/mm3 3.80 5.30	Hgb gm/dl 11.7 15.5	Hct % 36.0 46.0	MCV fl 80.0 99.0	MCH pg 25.0 31.0	MCHC gm/dl 32.0 34.	RDW % 11.5 14.5	Platelet 1000/ mm3 150.0 400.0	Gran % 43.0 70.0	Lymph % 22.0 41.0
ER 2220	15.6	3.69	11.5	33.3	90.3	31.2	34.5	17.3	290.0	90.4	5.9
Day 1 0750	11.7	3.22	10.2	29.3	91.0	31.6	34.7	17.5	243.0	***	***
Day 2 0400	9.1	31.12	9.8	28.8	92.3	31.3	33.9	17.4	201.0	83.2	10.4
Day 3 0230	8.6	2.64	8.4	24.6	93.4	32.0	34.3	18.5	176.0	78.0	12.0
Day 4 0350	8.3	2.72	8.6	25.4	93.5	31.7	33.8	18.6	217.0	78.9	13.7
Day 5 0423	3.9	2.55	8.0	23.9	93.5	31.5	33.6	18.4	197.0	76.5	15.2
Day 6 0400	6.7	2.79	8.7	26.2	93.7	31.3	33.3	18.0	232.0	73.7	16.7

Chemistry Results

Results: Units: Low Ref: High Ref:	Gluc. mg/dl 75 110	Na mEq/L 137 145	K+ mEq/L 3.6 5.0	Cl mEq/L 98.0 107.0	CO2 mEq/L 22.0 31.0	BUN mg/dl 9.0 21.0	Creat mg/dl 0.7 1.5	Ca++ mg/dl 8.4 11.5	Phos mg/dl 2.4 4.4	Mag mg/dl 1.4 1.8	Amyl U/L 30 110
ER2220	101	130	2.1	82.0	36.0	26.0	1.9	21.9	***	0.9	758
Day 1 0400	95	131	2.6	87	39	25.0	1.8	16.5	***	1.8	***
Day1 2100	93	139	3.5	100	32	23.0	1.7	18.4	0.9	1.4	***
Day 2 0400	91	141	3.9	106	27	21.0	1.7	15.4	2.7	2.3	229
Day 2 1800	137	141	3.3	110	22	17.0	1.5	13.8	1.1	***	***
Day 3 0230	105	141	4.1	111	22	16.0	1.4	12.5	3.2	1.2	190
Day 5 0400	91	142	4.4	116	20	7.0	1.4	9.4	2.2	0.8	***

Additional Labs

Results: Units: Low Ref: High Ref:	Lipase U/L 23 208	Troponin mg/ml <0.10	PT seconds 9.4 10.8	PTT seconds 24.0 31.0	Angiotensin Convert. Enzyme IU/L 9 67
ER2200	11732	0.42	11.3	<21	***
Day 1 1600	3717	0.34	***	***	***
Day 1 2100	2600	***	***	***	***
Day 2 0400	1630	***	***	***	22
Day 3 0230	1459	***	***	***	***

Urinalysis

Results: Units: Low Ref: High Ref:	Spec. Gravity 1.0005 1.0300	pH units 4.6 8.0	Leuk. #/ul Neg	Nitrites Neg	Protein mg/dl Neg	Gluc. mg/dl Norm	Ketones mg/dl Neg	Urobili Ehr./ U Neg	Bili mg/dl Neg	Blood /ul Neg
ER2250	1020	6.0	100	neg	30	norm	neg	norm	neg	10
Day 4 1700	1.005	7.0	neg	neg	neg	norm	neg	norm	neg	neg

Table 2: Common Causes of Hyperparathyroidism

- **Malignancy**
  - Tumors secreting PTH-related proteins
  - Ectopic production of Vitamin D substrates
  - Metastatic/lytic bone lesions
  - Hematologic cancers (myeloma, lymphomas, leukemia)
- **Endocrine**
  - Parathyroid disease (adenoma, hyperplasia, carcinoma)
  - Hyperthyroidism
  - Adrenal insufficiency
  - Pheochromocytoma
  - Multiple endocrine neoplasias (MEN 1 and MEN 2)
- **Granulomatous disease**
  - Tuberculosis
  - Sarcoidosis
  - Histoplasmosis
  - Coccidiomycosis
- **Drugs**
  - Lithium
  - Thiazides
  - Estrogen
  - Vitamin A
  - Vitamin D
- **Miscellaneous**
  - Immobilization
  - Milk alkali syndrome
  - Familial hypocalciuric hypercalcemia
  - Aluminum intoxication

Table 3: Clinical Manifestations of Hypercalcemia

Stones	Abdominal moans	Psychic groans
Nephrolithiasis Dehydration (impaired urinary concentration, decreased GFR, prerenal azotemia) Diabetes insipidus (polyuria polydipsia)	Nausea/ vomiting Pain Pancreatitis Anorexia Peptic ulcer disease Constipation	Memory loss Confusion Lethargy/coma Muscle weakness/fatigue
Bones		**Cardiovascular**
Arthritis/pain		Hypertension Short QT on ECG Cardiac arrhythmias/blocks

Progress During the Emergency Department Stay

Throughout her stay in the ED, the patient's blood pressure remained elevated despite administration of 0.1mg clonidine by mouth and 20mg diltiazem intravenously. Her mentation was consistently altered until after her admission to the Intensive Care Unit. A potassium chloride drip was initiated immediately after the panic value was discovered. Once potassium was completed, magnesium was initiated to be infused, as well as a fluid bolus of 0.9 normal saline followed by a maintenance rate of 100 ml/hr.

Diagnostic Considerations

The patient presented with altered mental status due to severe hypercalcemia. She also had acute pancreatitis secondary to the hypercalcemia and/or her chronic alcoholism, hypomagnesemia, and acute renal failure probably due to dehydration. The specific causes of hypercalcemia that need to be considered are in Table 2.

The clinical manifestations are often vague, affecting multiple organ systems. However, calcium levels greater than 14mg/dL associated with acute symptoms is considered critical and must be immediately addressed. Yet another mnemonic (stones, bones, abdominal moans, and psychic groans) will enable one to recall many of the signs and symptoms of hypercalcemia.

Outcome

The following morning, the patient was re-evaluated and found to have a myriad of symptoms associated with hypercalcemia. She complained of weakness, generalized abdominal pain, and severe constipation. Her cardiac monitor showed sinus bradycardia at 56 beats per minute. Calcitonin, magnesium, and phosphorus were initiated to resolve the electrolyte imbalances while a maintenance infusion of 0.9% normal saline provided a renal buffer. She remained hypertensive until the hypercalcemia resolved, at which time she was transferred from the ICU to a step-down telemetry unit. If warranted, patients in crisis may also be given loop diuretics, biphosphates, and hydrocortisone; other treatments will depend on specific causes. The patient was discharged home after six days with prescriptions for metoprolol extended release, amiodipine, oral phosphates, and magnesium oxide. She was instructed to discontinue the hydrochlorothiazide, which was probably an important exacerbating factor, and to follow-up with her primary care physician for further evaluation; abstinence from alcohol was also encouraged.

Loren Crown, M.D., is a clinical professor at the University of Tennessee. Currently, he is the medical advisor for the graduate training programs in emergency medicine in Jackson and Memphis.

Andra Kofahl, EMT-P, has 15 years of experience as an emergency room paramedic. Her undergraduate degree is in biotechnology. Ms. Kofahl was recently accepted to medical school.

Robert B. Smith, M.D., received an Emergency Medicine Fellowship at the University of Tennessee in 1998 and has been practicing emergency medicine for the past ten years. He currently works at four local hospitals in the St. Louis area. He is also a member of FEMA.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist.

Suggested Reading

1. Thomas P. Jacobs & John P. Bilezikian. "Rare causes of Hypercalcemia," The Journal of Clinical Endocrinology & Metabolism, Vol 90, No 11, 6316 – 6322, 2005.
2. Reinhard Ziegler. "Hypercalcemia Crisis," Journal of the American Society of Nephrology, J Am Soc Nephrol, 12:53-59, 2001.
3. Carroll, M., M.D., & Schade, D., M.D. (2003). A Practical Approach to Hypercalcemia.
4. Chishola, M, Pharm. D. & Taylor, T., Pharm. D. (1995). Acute Hypercalcemia. U.S. Pharmacist.
5. Harrison's Principles of Internal Medicine, 13th Edition. 1994.
6. Robin R. Hemphill, MD, MPH, "Hypercalcemia," emedicine, 2007.

Penetrating Eye Injury: A Case Study

Shane Havens, BS, M4
Omofolasade Kosoko-Lasaki, M.D., MSPH, MBA
Millicent Palmer, M.D.

Abstract

Eye injuries resulting from ocular trauma pose a frequent threat to vision the world over. The setting and causes of eye injury are diverse, but previous studies have demonstrated that the risk and type of injury is often correlated with age, gender, and race. While a focused history and prompt ocular examination are essential to immediate management, patient education regarding safety precautions and risk reduction help to prevent future recurrences.

This article describes a case of BB gun-related penetrating eye injury with a retained intraocular foreign body in a 10-year-old male. The case discussion will review the initial examination, resultant management decisions, and final visual outcome. A suggested approach to the management of patients with ocular trauma with emphasis on the differential diagnosis, clinical signs, management, and referral of common injuries to the eyes will be discussed.

Introduction

Vision is one of the most valued and powerful senses. Intact binocular vision plays an important role in development, independence, quality of life, and personal safety. Ocular injury is a frequent and preventable cause of visual impairment. The lifetime prevalence of sustaining any type of ocular trauma approaches 19.8%.[1] Eye injury covers a broad range of severity, from a small corneal epithelial abrasion to the more severe penetrating and globe rupture injuries.

The pediatric age group accounts for 8-14% of all eye injuries. Pediatric ocular injury is usually accidental and uniocular.[2,3] In contrast, adult ocular injury is usually the result of intentional assault. Males are more often affected than females, at a rate approximating 4:1, and suffer injury at a younger age.[1,4,5] Patients with a prior history of ocular trauma have a threefold increase in recurrence risk in a five-year period compared to those without prior injury.[1] Prompt evaluation and appropriate management of ocular injuries are paramount in the preservation of vision. Visual acuity, presence of an afferent pupillary defect, the type of injury, the location, and extent of penetrating injury, type of lens damage, presence and severity of vitreous hemorrhage, presence of and composition of intraocular foreign bodies mostly contribute to the final visual outcome in patients suffering penetrating ocular injuries.[6] In this article, we describe the evaluation and management of penetrating injury and intraocular BB foreign body in a 10-year-old male.

Case Report

M.R. is a 10-year-old male patient who presented to a local emergency department complaining of sudden severe left eye pain while shooting a BB gun with a friend without adult supervision. He reported the sudden onset of intense pain in the left eye after being struck by a BB that had ricocheted off a nearby tree. There were no complaints related to the right eye. He denied wearing any protective or prescription glasses at the time. He had no past history of ocular trauma, ocular surgery, glasses, or contact lens wear. Past medical history was unremarkable, and he was not on any medications. Current immunization for tetanus was confirmed by the parents. Family history was noncontributory. He lives at home with his parents and younger sister and attends elementary school.

On examination, M.R. was a well-developed 10-year-old male in acute ocular distress. On examination, his vision, using the Snellen visual acuity card at two feet, was 20/40 in the right eye and no light perception in the left with good illumination. The flashlight examination revealed no facial abnormalities. The right eye pupil was round, about 3.5 mm in diameter constricting to about 2.5 mm on direct light exam. There was no reversed afferent papillary defect. The pupil also reacted normally to accommodation. The left pupil was very difficult to evaluate due to the presence of blood in the anterior chamber. The left eye had normal eyelids without bruising, mild subconjunctival hemorrhage, mild conjunctival chemosis, and an irregular linear corneal laceration extending from seven to one o'clock. Examination of the left eye anterior chamber revealed

significant iris prolapse through the corneal laceration and hyphema. Visualization of the lens was obscured by the hyphema and necrotic iris tissue. Examination of the anterior and posterior segments of the right eye was normal without signs of trauma. Given the extent of the penetrating eye injury, the ocular examination was terminated, the eyes protected with a fox shield, and the patient was prepared for surgery. A pre-surgical computerized tomography (CT) of the orbits revealed a round, metallic foreign body at the level of the iris plane. Otherwise, all the other orbital structures were normal.

The diagnosis of penetrating eye injury with corneal laceration and intraocular metallic foreign body was made. The patient was given fentanyl for pain control, and informed consent was obtained from the parents. The patient was prepared for exam under general endotracheal anesthesia, repair of corneal laceration, and removal of intraocular foreign body.

In the operative suite the left eye was carefully prepped with 5% Betadine and a sterile eye drape was placed. Exam under anesthesia revealed a full thickness angled corneal laceration extending from corneal apex to seven o'clock approximately 5 mm and to 1 o'clock another 5 mm. Marked hyphema and an inferior displacement of the crystalline lens was observed on anterior chamber exam. Ten interrupted 10-0 nylon sutures were placed to reapproximate the corneal wound. The hemorrhage was aspirated from the anterior chamber using an irrigation-aspiration hand piece, and necrotic iris tissue was excised. A lensectomy was performed on the inferiorly displaced lens. Multiple attempts at extraction of the BB with BB forceps were unsuccessful. The BB was noted to move slightly posteriorly before attempts were abandoned, with the fear of further posterior migration and subsequent retinal damage. All superficial surgical wounds were closed with interrupted nylon sutures, and the wound was checked with a Fluorescein strip (Fluor-1-Strip, fluorescein sodium 1mg, Bausch & Lomb), and found to be watertight (Seidel negative). Subconjunctival injections, including 100 mg of cefazolin and dexamethasone 2 mg, were given inferiorly. At the end of the procedure topical 1% atropine prednisolone acetate 1% and Vigamox® (Moxifloxacin hydrochloride 0.5%, Alcon) were administered. Tobradex ophthalmic ointment (tobramycin and dexamethasone, Alcon) a double eye pad, and metal shield were applied to the left eye. He was then taken to the recovery area and later admitted in stable condition.

On hospital day two, the patient was taken back to the operative suite by a vitreoretinal specialist for removal of the BB, using a large intraocular magnet, open-sky anterior vitrectomy, revision of the corneal wound, and intravitreal antibiotic injection. The BB was successfully extracted using an external approach. The corneal wound was closed, and the anterior chamber was reformed. Intravitreal vancomycin (1 mg) and ceftazidime (2mg) were administered. Tobramycin/dexamethasone ophthalmic ointment, pressure patch, and a metal eye shield were placed over the left eye.

The patient was discharged from the hospital on day three with a clinic visit scheduled for the following day. His parents were advised to keep the metal eye shield in place at all times. Discharge eye medications included frequent administration of topical prednisolone acetate to control inflammation and prophylactic antibiotic coverage with Vigamox®. Tylenol with codeine was to be given as needed up to every six hours for pain and discomfort. The following day vision in the left eye remained at no light perception. The patient was followed closely over the next few days without improvement of visual acuity. After discussing the possibility of sympathetic ophthalmia, risks, benefits, and alternatives to surgical enucleation, in a blind eye, the family decided on enucleation and placement of prosthesis in the left socket. Polycarbonate eyeglasses were prescribed to protect the right eye from future injury.

Discussion

Ocular injury is classified into blunt or penetrating, depending on the causative factor. Blunt ocular trauma can be defined using the basic physics concept of energy exchange. Energy is transferred between the injurious object and periocular or globe structures without intrusion of the injured tissue by the offending object.[7] Penetrating injury involves intrusion into the injured tissue by the offending agent. Blunt trauma can produce a very different clinical scenario and pattern of tissue injury when compared to that of penetrating injury. Classifying the injury as blunt or penetrating early in the patient interaction will facilitate a more focused and effective exam, reducing the time required to provide definitive management. Initial evaluation and identification of penetrating ocular injuries is important in minimizing further ocular complications. Once the diagnosis of open globe is made, immediate referral for surgical exploration and repair should be made to the eye specialist. The eye should be protected with a fox metal eye shield while awaiting definitive treatment. Baseline exam findings and visual acuity are important in advising patients and family members on the prognosis and final visual outcomes. Findings supporting a favorable outcome (20/50 or better) include a normal lens at presentation and an anterior segment location of intraocular foreign body. However, more discouraging findings for final visual outcome, many present in the case being discussed, are afferent pupillary defect, visual acuity of light perception or, worse, prolapse of uveal tissue, BB injury, and foreign bodies posterior to the lens.[8] If there is a poor view of the fundus, one should consider B-scan ultrasonography to examine integrity of posterior tissues. Orbital radiographs or computed tomography (CT) scanning should be done if a foreign body may be present. Magnetic Resonance Imaging (MRI) is contraindicated in cases involving a suspected metallic foreign body. If a metallic intraocular foreign body is present, its prompt removal has been shown to be important in attaining the best visual outcome possible.[9] In the case that we presented, the decision to use an external approach to remove the BB was based on the greater strength of attraction observed between a BB of the same composition (copper lining with a steel core) and the large external magnet compared to that of the smaller internal magnet. A

series of 70 patients receiving treatment at McGill University Health Center in Quebec demonstrated that there was no significant difference in visual outcomes between external approaches to retrieval versus internal approaches (consisting of pars plana vitrectomy with removal using forceps or internal magnet).[10]

Antibiotics are generally started postoperatively in traumatic cases with open globes. Subconjunctival injections of vancomycin, ceftazidime or gentamicin, and dexamethasone postoperatively are recommended. Recommended topical antimicrobial therapy includes fourth generation fluoroquinolones, such as Vigamox® (moxifloxacin HCL 0.5) and Zymar (gatofloxacin 0.3%, Allergan) or, alternatively, fortified vancomycin and ceftazidime. Inflammation is managed with 1% prednisolone acetate ophthalmic solution. Cycloplegic agents, such as 1% atropine, twice daily will help in maintaining the depth of the anterior chamber and may enhance patients' comfort while facilitating the examination of the fundus. Broad spectrum intravenous antibiotics, like vancomycin, ceftazidime or fluoroquinolone, are commonly given preoperatively and discontinued after four to seven days.[11] The intravitreal injection of antibiotics has been shown to be an effective method of reducing the risk of post-traumatic endophthalmitis, a devastating intraocular infection.[12] The risk of endophthalmitis is increased in cases of ruptured globe injuries with or without retained intraocular foreign body. The most common organisms in post-traumatic endophthalmitis include Staphylococcus epidermidis and Bacillus species.

The Seidel test confirms or rules out cases of full thickness injuries to the anterior segment. In the Seidel test a moistened fluoroscein strip (Fluor-1-Strip, fluorescein sodium 1mg, Bausch & Lomb) is painted over the suspected injury site. The orange fluorescein changes to apple green if there is a wound leak, and a stream of aqueous can be observed with cobalt blue light.

Partial thickness corneal lacerations require topical broad-spectrum antibiotic coverage and a cycloplegic agent for relief of photophobia and pain associated with ciliary spasm. A bandage contact lens may also be helpful for structural or tectonic support. Full thickness corneal lacerations, as in the case presented, require surgical exploration and repair with postoperative administration of antibiotic and steroids. Thus, in all cases of penetrating ocular injury, close daily follow-up is required to monitor progress and identify any sign of complications. The patient with severe penetrating injuries should be followed closely for potential complications like endophthalmitis, recurrence of hyphema, ocular siderosis, and sympathetic opthalmia.[13] When considering pediatric enucleation, the prosthesis takes on special importance. Proper sizing and adjustments play a role in the growth and development of the orbit and facial symmetry, improving chances for an acceptable cosmetic appearance and avoiding potential future complications.

Epidemiology of Ocular Trauma

BB-related trauma is one of the most common causes of severe eye injury among adolescent males.[13] It is often the result of a ricocheted trajectory off a hard object near or behind the intended target. The muzzle velocity of projectiles fired from nonpowder guns ranges from 150 to 1200 feet per second and can maintain near maximum velocity after ricocheting off solid objects.[14]

In a review of National Injury Surveillance System data from 1993 to 2002, the rate of firearm-related eye injury was observed to decline while the rate of nonpowder (BB and pellet gun) injury remained relatively constant with a prevalence of approximately six per 1,000,000. Caucasian and Latino males were more likely to suffer nonpowder gun injuries, but African-American males more often sustained firearm-related eye injury.[15]

There is significant variability in the estimated number of eye injuries from all causes in the United States. The wide range of estimates can be attributed to the setting reporting the data. Inpatient facility reports tend to underestimate figures because eye injuries are not often severe enough to warrant hospitalization, while inpatient and outpatient (emergency department) reports neglect cases of ocular injury treated in private physician offices. The prevalence of emergency-department-treated eye injuries in the US is 3.15 per 1000 population. The highest rates are among men in their 20s and 30s and among those of American Indian and African-American descent. Most injuries are reported to occur in the home and are secondary to contusions or abrasions.[16,17] The prevalence increases to 6.98 per 1000 population when eye injuries that require treatment in inpatient, outpatient facilities, and private physician offices are considered. The more comprehensive approach used in this study also changed the demographics and most common modes of injury. Caucasian men in their 20s experienced the highest rates of ocular injury in this study population. The more common injury etiologies were superficial erosions, foreign bodies, contusions, and open wounds.[17,18]

Classifying Ocular Injury

Eye injury can be anatomically classified into anterior segment, posterior segment, adnexal, and orbital trauma. Ocular trauma can be described as penetrating or perforating. In a penetrating injury only one surface of an ocular structure is damaged (i.e. an entrance wound without an exit wound). Perforating injuries are defined as "double penetrating" injuries, creating both entry and exit wounds.[11] Blunt trauma can result in hematoma and contusion of periocular structures and globe ruptures. A rupture is a full thickness injury of the tissue that makes up the external boundaries of the globe. Ocular structures that are vulnerable to rupture injury include the limbus, areas of scleral thinning just posterior to rectus muscle insertions, and previous surgical incision sites.

The Ocular Trauma Classification Group provides a classification scheme for mechanical ocular trauma based upon the type and mechanism of injury, grade of injury based on visual acuity at presentation, and absence or presence of an afferent papillary defect.[17,19]

Management

A thorough history will help identify and detail the cause of injury. The history should include when, where, and how the injury occurred in the patient's own words. The reporting and documentation of the history and physical exam often have medicolegal consequences, as many are work-related or a result of assault. The physician should inquire into any previous treatments for the current injury and the events occurring from the time of injury until the time of presentation. Past medical history including eye injuries, ocular surgeries, or history of amblyopia, medications, allergies, family history, social history, and date of last tetanus vaccination complete the highlights of the eye injury history.

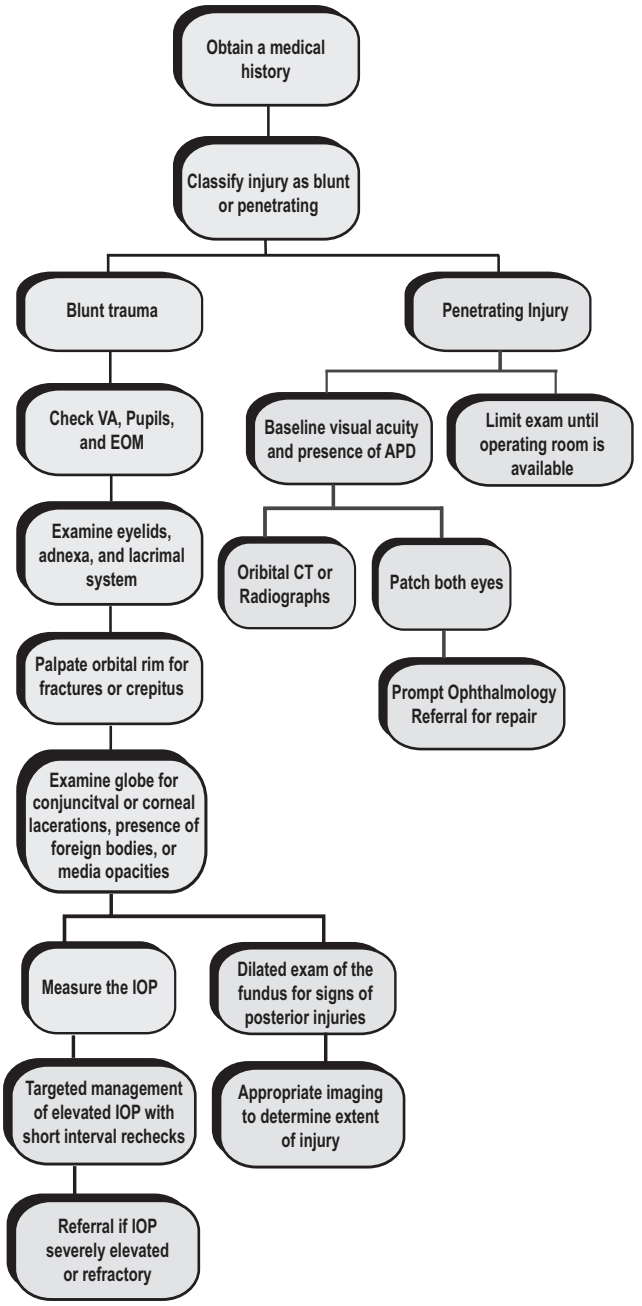


Figure 1: Management Guide for Ocular Trauma.
Adapted with Permission from J Rowe and O Kosoko-Lasaki.

The examination should start with an assessment of visual acuity. Snellen Visual Acuity chart or near card testing are standard, but newspaper print is acceptable if neither is available. Counting fingers, hand movement, light perception, or no light perception should be documented, if the patient is unable to read the largest line (or 20/400 equivalent) on the visual acuity chart.

The examination of the eye should be comprehensive and must follow a logical sequence. A common practice among eye specialists is to examine the eye from the external and anterior structures to those more posterior. Examination of the traumatized eye thus begins with examination of the external structures and bony orbit. Palpation of the orbital rim detects fractures and or crepitus. The eyelids are evaluated for laceration, noting any involvement of the lid margins or lacrimal system. Examination of the pupils for their shape, size, symmetry, direct, and consensual responses to light should be noted. The pupillary exam is especially important in cases where intracranial injury is possible. The pupils should not be pharmacologically dilated until mental status has been assessed and neurologic status deemed stable. Evaluation of the extraocular muscle function in all directions of gaze may identify a restriction of gaze attributable to muscle entrapment within an orbital fracture or traumatic cranial nerve injury. Slit lamp examination of the globe allows classification of conjunctival hyperemia, subconjunctival hemorrhage, hyphema, corneal opacities and edema, foreign bodies, traumatic iris tears, and lens location and stability. Fluorescein strips can be used to delineate corneal abrasions and to detect any active aqueous leakage from the anterior chamber using the Seidel test. Intraocular pressure should be measured in all cases of ocular trauma, except those with an obvious open-globe injury, in which case manipulation of the globe should be minimized until evaluation and surgical repair is considered by an ophthalmologist.

Dilated fundus exam provides a view of the retina, macula, optic disc, and ocular vasculature. The red reflex is evaluated for brightness, shape, and symmetry between the two eyes. The ocular media is then checked for opacities, foreign body, or hemorrhage. Finally, the retina is evaluated for signs of choroidal rupture, commotio retinae, subretinal hemorrhage, and traumatic retinal tears and detachments. Depending on the type of injury, various forms of imaging modalities may be pursued. Radiographs, ultrasonography, CT, or MRI may be ordered. MRI is always contraindicated when a metallic foreign body is suspected. If perforating injury is suspected, imaging should be performed, both eyes patched, and immediate ophthalmology referral made for further evaluation and probable surgical repair. Figure 1 provides a general management model for blunt and penetrating ocular trauma.

Injuries to Anterior Structures

Orbital Injury: Trauma to the orbit, like all of ocular trauma, can be classified as blunt or penetrating. Orbital contusion is periocular bruising secondary to blunt trauma that can present with associated injury to the globe, sinuses, and bony orbit. The presence of an afferent pupillary defect, confrontation vi-

sual field defect, limited extraocular motility, and proptosis are ominous signs that should be further evaluated. Ice compresses can be used for 48 hours after injury to limit the swelling if the globe is found to be intact and vision is not affected. Orbital compartment syndrome is caused by the accumulation of blood from a retrobulbar hemorrhage. Clinical signs of orbital compartment syndrome include a tense orbit, proptosis, resistance to retropulsion of the globe, and limitation of eye movements. Emergency ophthalmology referral for possible lateral canthotomy is warranted.

Fractures of the orbit most commonly involve the orbital floor, and are termed blow-out fractures. The fracture of the maxillary bone allows prolapse of ocular contents into the maxillary sinus. Signs include diplopia in up or down gaze, enophthalmos, and orbital and eyelid emphysema if the patient has blown his or her nose since the injury. Urgent surgery is indicated in pediatric cases with signs of rectus muscle entrapment, but can be scheduled one week after injury in adults to allow reduction of swelling in the meantime.

Eyelid Injury: Eyelid trauma may often appear trivial but can have profound effects on injury associated morbidity. Recovery of full eyelid function and maintenance of the lacrimal apparatus are important considerations when approaching lid trauma. An eyelid contusion consists of edema and ecchymosis that often follows blunt trauma. The hematoma is embedded within the layers of the orbicularis oculi muscle and cannot be surgically evacuated. The hematoma of eyelid contusions does not typically result in ocular compartment syndrome, because they are located anterior to the orbital septum. Traumatic ptosis can result from direct injury to the levator muscles or mechanical effects of edema and hematoma and can take months to resolve. Cool compresses for 10-15 minutes a few times a day are generally sufficient in cases of isolated lid contusions.

When presented with eyelid avulsion (tearing or shearing injury) or laceration, the restoration of any lacrimal injury should be considered prior to repair. Injury to the canalicular system may require silicone tube stenting prior to careful suture placement. Unrecognized punctal, canalicular, or lacrimal sac injuries that are sutured without the use of a stent can lead to a lacrimal drainage obstruction and, subsequently, a chronic tearing state. Injuries to the inferior punctum, inferior canaliculus, or common canaliculus are more likely to cause significant tearing. Referral to an ophthalmologist is mandatory in cases of canalicular injury. The presence of orbital fat in the wound indicates a likely violation of the orbital septum and should be followed accordingly. Contaminated wounds should be debrided and treated with appropriate systemic and topical antibiotics. Tetanus vaccination status should be considered in all patients with these types of injuries.

Cornea and Conjunctival Injury: The anterior location of the cornea and conjunctiva make the structures vulnerable to both blunt and penetrating trauma. The conjunctiva may be torn or lacerated without damage to deeper or adjacent tissue. An area of hyperemia often surrounds the injury, which can be better visualized using fluoroscein. It is important to rule out damage to the underlying sclera and to rule out the presence of a foreign body. Smaller lacerations, generally those less than 5 mm in diameter, will heal without surgical intervention. Lacerations larger than 5 mm have the best outcomes when sutures are placed. Topical broad-spectrum antibiotic coverage is applied to prevent secondary infection of exposed tissues.

Corneal abrasions consist of epithelial cell erosions with an intact Bowman's membrane. The patient with a corneal abrasion often complains of a painful eye with reflex blepharospasm (involuntary lid squeezing) and increased tearing from the affected eye. The defect should be examined under the slit lamp to assess the size, location in relation to the visual axis, and rule out the presence of a foreign body. Application of fluoroscein is used to visualize the defect, using a cobalt blue light. The benefit of patching the eye is controversial; however, it is a common practice by most health care providers. Close follow-up of the patient until the corneal epithelium has completely healed is standard practice.[11]

Penetrating injuries of the cornea also require a complete eye examination to rule out damage to intraocular structures and the presence of a foreign body despite the obvious nature of the injury. Prolapsed iris tissue may be repositioned or removed during surgery, depending on the duration of exposure or the state (necrotic or not) of the tissue. Small puncture wounds and shelved lacerations often heal without suturing. Bandage contact lens and patching are options for treatment under the close care of an ophthalmologist. It is important to remove all foreign material and to reform the anterior chamber before placing corneal sutures.

Rupture of the cornea and/or sclera can result from blunt trauma. Susceptible sites of rupture include the limbus, posterior to the rectus muscle insertions, and areas of previous surgical incision sites. Signs of rupture include subconjunctival hemorrhage, hyphema, a change in anterior chamber depth, limited extraocular movements in the direction of gaze towards the rupture, and low intraocular pressure (hypotony). Globe rupture is a surgical emergency. The patient should be referred to ophthalmology for surgical evaluation and treatment.

Hyphema is blood in the anterior chamber, one of the signs of severe blunt or penetrating trauma. Partial or microscopic hyphema will resolve spontaneously through macrophage phagocytosis and via the aqueous drainage system.[11] Hemorrhage in the anterior chamber can occur up to two weeks after the initial trauma event. If large, the hemorrhage may cause an increase in intraocular pressure and blood staining of the corneal endothelium. Corneal blood staining can occur rapidly but takes months to completely clear and may require anterior chamber washout. Most cases of hyphema are managed conservatively by advising the patient to wear dark sunglasses for comfort. In African-Americans, it is important to avoid the use of carbonic anhydrase inhibitors in the lowering of the intraocular pressure because of the risk of sickling of the red blood cells in those at risk.[11] Surgery is indicated if there is blood staining of the cor-

neal endothelium or in severe medically uncontrolled intraocular pressure elevation.[11]

Uveal Injury: Injury and damage to the anterior iris and cilary body can occur with trauma. Traumatic iritis is an inflammation of the iris tissue itself with associated hyperemia (perilimbal), pain, photophobia, and tearing. The traumatic event may cause tears in the pupillary sphincter muscles. These tears are seen as irregular areas in the pupillary ruff. Pupillary constriction may be limited by the defects in these muscle fibers resulting in traumatic mydriasis. Treatment of traumatic iritis consists of pharmacologic mydriasis for comfort and topical steroids to control the inflammation. Iridodialysis is the tearing of the iris root from the ciliary body. The tear is permanent, and surgical treatment is not needed but may be considered in severe cases. Traumatic iritis may result in the formation of anterior or posterior synechiae, iris adhesions to the cornea, and lens capsule respectively.

Lens Injury: Trauma to the lens may leave a ring of pigment, a Vossius ring, on the anterior lens capsule. This finding is benign and usually clears with time but, in some cases, can permanently stain the capsule. Ocular trauma may also result in lens subluxation or dislocation. Anterior displacement of the lens may result in pupillary block glaucoma. Trauma to the lens may also result in premature cataract formation years after the inciting injury.

Glaucoma Secondary to Trauma

Angle recession is a tear between the longitudinal and circular muscles of the ciliary body and is the most common complication of blunt ocular trauma, occurring in 80.5% of cases following blunt ocular trauma.[17,20,21] Ten percent of patients with angle recession that involves more than 2/3 of the angle develop glaucoma secondary to scarring of angle structures. The damage from angle recession is seen as a widening of the ciliary band on gonioscopy. Treatment involves topical and, in some cases, oral medications to reduce a high intraocular pressure in the days following the trauma. Patients with significant angle recession injuries should be followed by an ophthalmologist for IOP elevations, visual field testing, and optic nerve changes.[11] Penetrating injury can also induce elevated intraocular pressures and subsequent glaucomatous changes of the optic nerve. A recent review of the United States Eye Injury Registry showed that 2.64% of patients suffering penetrating ocular trauma develop glaucoma.[17,21] A dislocated lens may also block flow through the trabecular meshwork. Risk factors that have been shown to be associated with post-traumatic glaucoma include: advanced age, lens injury, inflammation of the anterior chamber, and a baseline visual acuity of 20/200.[20]

Intraocular foreign bodies can cause mechanical and other tissue alterations that may result in secondary glaucoma. Intraocular metals cause characteristic late tissue alterations. For example, iron-containing materials can cause ocular siderosis, a staining of the trabecular meshwork, and toxicity to other angle structures. Copper can lead to chalcosis with similar toxicity to structures of the iridocorneal angle if left in place. These changes make identification and removal of foreign bodies essential to minimizing late tissue alterations and the development of secondary glaucoma. The viable red blood cells, degenerated red blood cells, and macrophages containing ingested red blood cell remnants that accompany hyphema can also obstruct the trabecular meshwork.

Treatment of post-traumatic glaucoma is aimed at reducing IOP by decreasing aqueous formation by the ciliary body, preventing further obstruction to aqueous outflow, or creating an alternate aqueous drainage route. An aqueous washout procedure can be used in cases of uncontrolled IOP elevation related to hemorrhage.[11] Ophthalmic preparations of prostaglandin analogues, beta-adrenergic receptor antagonists, alpha-adrenergic receptor agonists, and carbonic anhydrase inhibitors are commonly used to help reduce IOP. In acute or refractory cases, oral carbonic anhydrase inhibitors or osmotic diuretics can be utilized. In addition, surgical options should be considered in cases of elevated IOP that is not controlled with medical management. Potential surgically therapeutic options include trabeculectomy with anti-metabolites or implantation of a glaucoma Seton valve.

Injury to Posterior Structures

Trauma may manifest in the posterior segment as commotio retinae, choroidal rupture, posterior scleral rupture, or retinal breaks and detachments. Commotio retinae generally presents as a gray-white discoloration of the retina secondary to disruption of the outer segment of the photoreceptors. It may be accompanied by choroidal rupture and subretinal hemorrhage and can be observed initially as asymmetry of the red reflex in the injured eye if sufficiently large and located on the central posterior pole. Choroidal rupture is a tear in the choroid, Bruch's membrane (the layer between the choroid and retinal pigment epithelium), and the retinal pigment epithelium following blunt trauma. These findings on dilated exam are supplemented by findings on ultrasound, CT scan, and MRI. Traumatic retinal detachments require emergent evaluation and possible repair by an ophthalmologist.[11]

Sympathetic Ophthalmia: Sympathetic ophthalmia is the most feared complication of ocular trauma. It is characterized by a bilateral, granulomatous uveitis following penetrating eye injury. The pathophysiology is believed to involve an autoimmune reaction to uveal antigens encountered as a result of the initial trauma event. Inflammation first occurs in the injured eye. The uninjured eye is termed the "sympathizing" eye and becomes secondarily affected. Symptoms include blurred vision, photophobia, pain, and ultimately blindness. Once established, sympathetic opthalmia requires steroids and immunosuppressant agents to slow the progression. Onset can occur anytime from one week to several years following the injury, but most cases do not occur before two weeks after injury. For this reason, injured eyes that have little or no potential for visual recovery are strongly considered for enucleation within two weeks after injury. In the case presented in this article the eye had no light perception and was recommended for enucleation.

Conclusion

Penetrating eye injuries can be challenging to assess, given the associated pain and difficulty with examination. A good history and minimal examination will prompt referral for management of these serious injuries.

While surgical treatment modalities continue to improve, the most effective approach to ocular trauma is a proactive approach to prevention. Primary prevention of ocular injury is firmly based in improving patient education, proper safety equipment and practices, risk reduction, and close supervision of all high-risk activities. Prevention of ocular trauma, like all types of trauma, remains an ongoing battle. Legislation efforts regarding nonpowder or air guns are in place or underway in 30 states. New York has the strictest regulations in the US. In New York City BB guns are entirely prohibited and licenses are not available. In the rest of the state, no one under the age of 16 can purchase or use a pellet gun unsupervised. Florida has also enacted legislation aimed at reducing injury rates. Florida state law holds a minor under 16 years of age to be guilty of a second-degree misdemeanor if found using a BB gun without adult supervision. The laws vary from state to state, with some states regarding nonpowder guns as firearms and other not addressing them at all. Many authors support wider restrictions on the sale and use of nonpowder guns.[14] Others promote improving safety education and requiring proper apparel while using nonpowder guns. Standardization of warning labels and their strategic placement has been considered and modified. Perhaps a short safety course and test can be included in store packaging to be administered by a supervisory adult prior to use. Whatever means of prevention is pursued, the goal remains to minimize the risk of injury via responsible operation and safety precautions.

Finally, we recommend that all patients with previous ocular injury should be fitted with protective (polycarbonate) glasses, especially if they are involved in active sport activities.

Shane Havens, B.S., will graduate from the Creighton University School of Medicine in May 2009 and will pursue his residency training in ophthalmology. He is an active volunteer with Sight Savers, Glaucoma Caucus Initiative, Body Basics, and Prevent Blindness Nebraska.

Omofolasade Kosoki-Lasaki, M.D., Board Certified in Ophthalmology, is Associate Vice President, Health Sciences, and Professor of Surgery (Ophthalmology), Preventive Medicine, and Public Health at Creighton University, Omaha, NE.

Millicent Palmer, M.D., Chief of Ophthalmology for the Nebraska Western Iowa Health Care System, oversees ophthalmology services for the Omaha VA as well as Lincoln and Grand Island. Dr. Palmer, an Associate Professor of Surgery at Creighton University Medical Center, has played an important role in Glaucoma Awareness and Screening Initiatives.

The authors would like to thank Jess Boysen, M.D., Resident of University of Nebraska Medical Center, and Thomas Hejkal, MD, Attending Physician and Associate Professor of Ophthalmology, University of Nebraska Medical Center, for sharing the use of the case presented.

The authors would like to thank Reba Donahue for her editorial assistance in preparing this document.

Potential Financial Conflicts of Interest: By AJCM policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest. The authors have stated that no such relationships exist.

References

1. T Wong, BE Dlein, R Klein. The prevalence and 5-year incidence of ocular trauma. The Beaver Dam Eye Study. Ophthalmology 107(12): 2196-202.
2. PV Scribano, M Nance, P Reilly et al. Paediatric nonpowder firearm injuries: Outcomes in an urban paediatric setting. Paediatrics 1997; 100:E5.
3. JV Takvam, A Midelfort. Survey of eye injuries in Norwegian children. Acta Ophthalmol (Copenh) 1993; 71:500-5.
4. AL Dannenberg, LM Parver, RJ Brechner, L Khoo. Penetration eye injuries in the workplace. The National Eye Trauma System Registry. Arch Ophthalmol 110: 843-848.
5. AL Dannenberg, LM Parver, C J Fowler. Penetrating eye injuries related to assault. The National Eye Trauma System Registry. Arch Ophthalmol 110: 849-852.
6. E DeJuan, P Sterneberg, RG Michels. Penetrating ocular injuries. Types of injuries and visual results. Ophthalmology 1983; 90:1318-22.
7. M Forsyth. Blunt Trauma. Washington State Department of Health Presentation. www.doh.wa.gov/hsqa/emstrauma/OTEP/blunttrauma.ppt. Accessed 8/30/08.
8. JP Ehlers, DY Kunimoto, S Ittoop et al. Metallic Intraocular Foreign Bodies: Characteristics, Interventions, and Prognostic Factors for Visual Outcome and Globe Survival. Am J Ophthalmol. 2008 Jul 7.
9. M Soheilian, A Abolhasani, H Ahmadieh et al. Management of magnetic intravitreal foreign bodies in 71 eyes. Ophthalmic Surg Lasers Imaging. 2004 Sep-Oct;35(5):372-8.
10. DR Chow, BR Garretson, B Kuczynski et al. External versus internal approach to the removal of metallic intraocular foreign bodies. Retina. 2000;20(4):364-9.
11. P Kaiser, N Freidman, R Pineda. The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology, 2nd Edition. Saunders, Philadelphia PA, 2004. 112-13 and 152-153.
12. M Soheilian, N Rafati, M Mohebbi, et al. Prophylaxis of Acute Posttraumatic Bacterial Endophthalmitis: A Multicenter, Randomized Clinical Trial of Intraocular Antibiotic Injection, Report 2. Arch Ophthalmol 125: 460-465.
13. Newman TL, Russo PA. Ocular sequelae of BB injuries to the eye and surrounding adnexa. J Am Optom Assoc. 1998 Sep;69(9):583-90.
14. D Laraque and the Committee on Injury, Violence, and Poison Prevention. Injury Risk of Nonpowder Guns. Pediatrics Vol. 114 No.5: 1357-61.
15. McGwin G Jr, Hall TA, Xie A, Owsley C. Gun-related eye injury in the United States, 1993-2002. Ophthalmic Epidemiol. 2006 Feb;13(1):15-21.
16. G McGwin Jr, C Owsley. Incidence of Emergency Department–Treated Eye Injury in the United States. Arch Ophthalmol 123: 662-666.
17. J Rowe and O Kosoko-Lasaki. Review of ocular trauma. Archives of Ibadan Medicine, Vol 7, 1: 47-50.

18. G McGwin Jr, A Xie, C Owsley. Rate of Eye Injury in the United States. Arch Ophthalmol 123: 970-976.

19. DJ Pieramici, JP Sternberg, TM Aaberg et al. The Ocular Trauma Classification Group. A system for classifying mechanical injuries of the eye (globe). American Journal of Ophthalmology 1997; 123: 820-31.

20. YM Canavan, DB Archer. Anterior segment consequences of blunt ocular injury. British Journal of Ophthalmology 1982' 66:549-55.

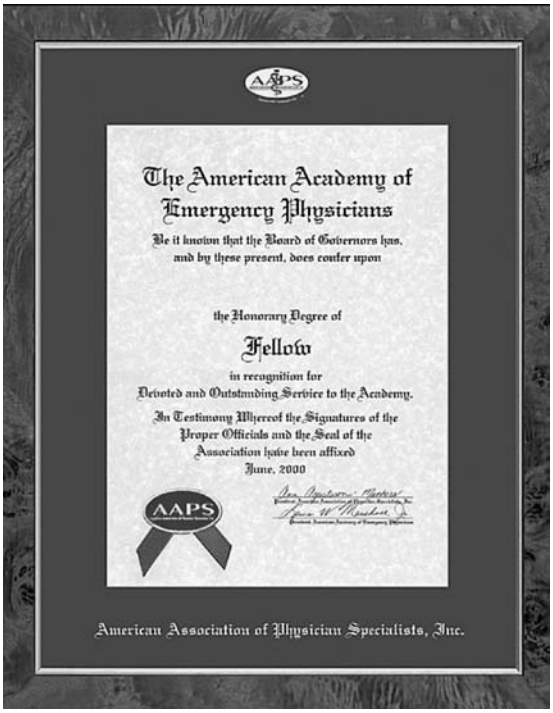
21. CA Girken, G McGwin, R Morris, R Kuhn. Glaucoma following penetrating ocular trauma: a cohort study of the United States Eye Injury Registry. American Journal of Ophthalmology 2005 Jan; 139: 100-05.

The Distinguished Degree of Fellow

The American Association of Physician Specialists, Inc. (AAPS), Recognizes Its Leaders

The Academies of Medicine of the American Association of Physician Specialists, Inc., awards the Degree of Fellow to those members who have demonstrated leadership and participation within their Academies by meeting specific criteria.

Do You Qualify?



If you meet these criteria, consider submitting a completed application to your Academy president by December 31 of each year for consideration for the following year.

- Initial certification received – minimum of two years
- Member of an Academy – minimum of three years
- Registered and attended three AAPS Annual Scientific Meetings within past five years
- Fulfill three of the following four criteria:

  ➢ Lectured or presented a poster at an AAPS Annual Scientific Meeting

  ➢ Published an article in the *American Journal of Clinical Medicine (AJCM)*

  ➢ Served as Academy of Medicine Officer, Governor, or Board of Certification Member, or AAPS Committee Chair

  ➢ Served as Oral Examiner, member of Examination Committee, or participated in Certification Workshop for the Board of Certification examination procedures

AAPS
AMERICAN ASSOCIATION OF PHYSICIAN SPECIALISTS, INC.

If you are awarded the Degree of Fellow, you must attend the Annual Scientific Meeting to receive the award.

You may download the Fellow application at www.aapsus.org or request an application by contacting the Executive Office at 813-433-2277.

The American Association of Physician Specialists, Inc., and the American Board of Physician Specialties are proud to introduce our staff and headquarters.

EXECUTIVE DEPARTMENT

Responsible for management and operations of Executive Committee, Board of Directors, Academies of Medicine, House of Delegates, Past Presidents, Awards, and Degree of Fellow

William J. Carbone, CEO

Nadine B. Simone, Executive Assistant

GOVERNMENTAL AFFAIRS

Responsible for State and Federal Legislation, Legislative and Recognition Issues, Medical Mission Outlook

Timothy J. Bell, Director of Governmental Affairs

Lauren E. Withrow, Governmental Affairs Coordinator

CME, MEETINGS, RECRUITMENT & RETENTION

Responsible for Continuing Medical Education, Meeting Planning and Management, Recruitment and Retention, Publications, Auxiliary

Esther L. Berg, Director of CME, Meetings, and Recruitment & Retention

Keely M. Clarke, CME, Meetings, and Recruitment & Retention Coordinator

CERTIFICATION DEPARTMENT

Responsible for all matters pertaining to Certification including Initial Inquiries, Requirements, Recertification, Boards of Certification, Examination Information

Cassandra R. Newby, Director of Certification

Susan C. LoBianco, Certification Coordinator

Theresa R. Rodriguez, Certification Coordinator

Marilyn D. Whitfield, Certification Coordinator

FINANCE & OPERATIONS

Responsible for Dues, Billing and Payments, Facilities, and Personnel

Anthony J. Durante, Director of Finance and Operations

Georgine C. Wasser, Finance & Operations Coordinator

Debi S. Colmorgen, Communications Coordinator

We encourage you to let us assist you with association matters – large and small. We welcome your ideas and suggestions. Don't hesitate to call on your AAPS Team.

UPCOMING EPIDEMICS

THEY'RE NOT JUST INFECTIONS

2009 HOUSE OF DELEGATES & ANNUAL SCIENTIFIC MEETING




SAN DIEGO, CALIFORNIA

June 22–27, 2009

EARLY REGISTRATION RATES!

REGISTER BY APRIL 30, 2009 TO SAVE $$

MAKE YOUR HOTEL RESERVATIONS NOW!

AAPS
AMERICAN ASSOCIATION OF PHYSICIAN SPECIALISTS, INC.

5550 West Executive Drive
Suite 400
Tampa, Florida 33609-1035

PRESORTED
STANDARD
U. S. POSTAGE PAID
PERMIT # 2483
TAMPA, FL