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ACEPNow

n light of ACEP's approaching golden anniversary, ACEP Now took a look back at the last 50 years of emergency medicine and the people and trends that have shaped the specialty. ACEP Now Medical Editor-in-Chief Kevin Klauer,

DO, EJD, FACEP, recently discussed the history of the specialty with two emergency physicians who have been members of the same EM group for their entire 45-year careers, a past ACEP President, and one of the next generation of EM leaders. The group discussed the origins of emergency medicine, how it has grown and changed over the years, and what the future holds. Here are some highlights from that conversation.

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Women's sabre team at the Zonal Championships, where they won gold medals. The team won bronze medals at the Rio Olympics. Left to right: Dagmara Wozniak, Ibtihaj Muhammad, Ann Marsh-Senic, Monika Aksamit, and Mariel Zagun



CORAL! Lower extremity weakness caused by an aquarium decoration

by WENXIA ZHAO, MD, AND J. MICHAEL BALLESTER, MD

oanthid corals are commonly kept in home aquariums. They produce a potent palytoxin that has various neurologic effects, including paresthesia,

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sion. We report on the case of a previously healthy male http://www.acep.org/ who handled a zo-ACEPeCME/ TO COMPLETE THE anthid coral in his ACTIVITY AND EARN aquarium and had FREE AMA PRA CATEGORY 1 CREDIT. subsequent bilateral lower extremity weakness. His

weakness, and res-

piratory depres-

symptoms resolved after supportive treatment of oral potassium replacement and intravenous fluids.

The Case

A previously healthy 34-year-old male presented to the emergency department with bilateral lower extremity weakness. He was able to walk when he woke up that morning but then was unable to stand without support. The day before presentation, the patient was moving a zoanthid coral with his bare hands. He denied any injury from the coral, but he had superficial lacerations on **CONTINUED** on page 5

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OPINIONS FROM EMERGENCY MEDICINE

A NEW SPIN



CULTURAL SENSITIVITY IN EMERGENCY MEDICINE: THE CONVERSATION CONTINUES

Black MD

ACEP's steps to promote diversity are crucial to supporting the needs of emergency physicians

ightarrow BY JENICE FORDE-BAKER, MD, FACEP \leftarrow

IT WAS MY USUAL DAY IN THE EMERGENCY DEPARTMENT at a busy community hospital with 55,000 annual visits. Midway through my shift of seeing abdominal pain, headaches, and febrile infants, the medic phone rang. They were bringing in an altered elderly patient with fever and low blood pressure. The medics arrived, and as I walked into the room, my team was already placing IVs, attaching monitors, and taking vital signs. I introduced myself as Dr. Forde-Baker, and the patient said, "Get away from me you...you...black person!" with disdain dripping from her voice.

Time stopped. I was instantly flooded with memories of similar encounters of hate and prejudice because of melanin: having to state which Ivy League schools I attended and how long I've been in practice; being asked if I am really the assistant director of the department; and having to remind patients and family members over and over that I am, in fact, the physician who has been caring for them for the past three to four hours.

Only a second had passed. I breathed, and I explained that I am her doctor and will be caring for her. My team supported me and informed the patient that she was getting the best care possible, and she did. This patient was treated with the same high quality of care I provide all of my patients, but inside, I felt disrespected, enraged, and diminished.

I know that I am not alone. In 1864, Rebecca Lee Crumpler was the first and only black woman

who earned a medical degree from the New England Female Medical College. Undoubtedly, she faced overt racism from male colleagues, pharmacists who would not fill her medications, and people who guipped that "MD" stood for "mule driver." Now, 154 years later, African-Americans only make up 6.3 percent of American physicians, of which, 55 percent are black women. And 154 years later, we are still facing racism.

Today, there is a history of distrust in the African-American community due to the exploitation of black people in medical research. Tuskegee syphilis trials and Dr. James Marion Sims gynecological surgeries are a few of the documented cases of heinous abuse of black people performed in the name of medical research. This has lead to a culture of fear, distrust, and



health disparities due to race and economics. Obesity, diabetes, and hypertension are a few of the medical illnesses disproportionately affecting black Americans. I contend that this is also due to a sense of isolation that African-American patients feel in the medical community. As a black female physician, I understand that feeling. Although I am a member of the group of more than 35,000 emergency physicians in the specialty, I

> am also a black woman. My appearance enters the exam room before I can utter a word. Most times, I am welcomed and embark on the physician-patient relationship. Sometimes, I am met with hate and feel belittled and burdened because the duty to provide care is still paramount and supersedes all else.

> As a black female physician, my experiences with patients are different from those of my white male counterparts. Increasing

diversity and recognizing the varied experiences physicians face are crucial to the wellness and feeling of inclusion of all emergency physicians. ACEP is taking that step to promote diversity and cultural awareness. Our President, Dr. Rebecca Parker, is making cultural diversity a part of her platform. The recent summit on diversity is the first step in having a real conversation about diversity in medicine from the physician's perspective. ACEP must hear from diverse physicians to acknowledge our experiences and support our needs. O



DR. FORDE-BAKER is assistant director of emergency medicine at Our Lady of Lourdes Medical Center in Camden, New Jersey.

"A New Spin" is the personal perspective of the author and does not represent an official position of ACEP Now or ACEP.

Looking Forward: How Emergency Departments Can Embrace Diversity and Inclusion

BY SHERYL HERON, MD, MPH, FACEP

n response to the article "Black MD" by Jenice Forde-Baker, MD, FACEP, the need to prove one's ability and capability as a physician in certain patient encounters resonated with me as an African-American woman physician and, I am sure, other minority groups. The author



speaks of her painful dismissal as a black woman and her need to justify her credentials. These realities are not new, given the history of African-Americans and other racial, ethnic, and other minority groupssuch as the Tuskegee study of untreated syphilis in black men noted by the author and the more recent story of Henrietta Lacks, a poor black tobacco famer whose cells, known as HeLa cells, were used without her knowledge for developing the polio vaccine. The patient-physician encounter is a sacred

Dr. Heron at a signing for Diversity and Inclusion in Quality Patient Care at ACEP16.

trust, and the patient's clinical experiences influence their health care outcomes and their trust in the medical system. The author speaks of the isolation felt as a physician of color compared to her other colleagues and the negative impact on her wellness from her unpleasant patient encounter. How can we address these challenges and create a health care environment and culture that is welcoming to all and intolerant of these insults? In these ever-changing times, the urgency and need to address individual and systemic racism are essential.

In her article, the author commends ACEP President Rebecca Parker, MD, for ensuring cultural diversity is a part of ACEP's priorities. This has been demonstrated by Dr. Parker's creation of a Diversity and Inclusion Task Force to address this critical and timely topic. However, to achieve success, we should ask ourselves, what can our respective institutions do to ensure that there is a welcoming environment that celebrates diversity and inclusion? The Association of American Medical Colleges' lens toward diversity, inclusion, and health equity as a driver for excellence is one example. Each institution or health care system can commit to the concept of diversity as drivers for excellence by 1) creating a strategic plan to address diversity and inclusion, 2) committing to leadership and mentoring opportunities for underrepresented groups, and 3) participating in cultural competency education and unconscious bias training. Most important, to achieve success in creating a culture that celebrates diversity and welcomes inclusion, there must be commitment by the leadership of your organization and/or institution. Dr. Parker has demonstrated that leadership. An honest reflection of your working environment and a commitment to open dialogue Baker concludes, "ACEP must hear from diverse physicians to acknowledge our experiences and support our needs." Are you willing to do the same? The work may be difficult; however, the efforts and rewards will be well worth it. .

DR. HERON is professor of emergency medicine and assistant dean at Emory University School of Medicine in Atlanta; a mem-ber of the ACEP Diversity and Inclusion Task Force; and an editor of Diversity and Inclusion in Quality Patient Care, the first textbook in emergency medicine addressing diversity and inclusion.

DON'T TOUCH THE CORAL! | CONTINUED FROM PAGE 1

his hands from yard work. He denied tick exposure, camping, pets, recent travel, fever, illness, previous medical history, and personal/ family history of neuromuscular or rheumatologic disorders. His physical exam showed inability to flex his bilateral quadriceps, with 4/5 strength on plantar and dorsiflexion of ankles and knees. He had equal sensation bilaterally, including the anterior thighs. There were no obvious injuries. CT of the brain and cervical spine were negative. Labs showed a potassium of 2.7. He was admitted to the hospital following a negative MRI. He received oral potassium and intravenous fluids with resolution of his weakness. Neurology suspected hypokalemic periodic paralysis (HPP). The patient was discharged with neurology follow-up.

Discussion

Zoanthid corals are often kept in home aquariums and produce a deadly vasoconstrictor called palytoxin. The toxin binds Na+/K+ AT-Pase and destroys the ion gradient across cell membranes, leading to cell death. Doses as low as 0.033 mcg/kg can be lethal. Case reports and anecdotal forums show that most exposures come from inhalation or skin contact. There are currently no guidelines for proper protective gear for handling these corals. There is no known antidote, and treatment is supportive.1

A 2015 Morbidity and Mortality Weekly

"Doses as low as 0.033 mcg/kg can be lethal. Case reports and anecdotal forums show that most exposures come from inhalation or skin contact. There are currently no guidelines for proper protective gear for handling these corals. There is no known antidote and treatment is supportive."

Report from the Centers for Disease Control and Prevention shows that palytoxin exposure may cause nausea, headache, renal failure, weakness, ataxia, fever, and other symptoms.² These cases were seen in Alaskan aquarium shops and homes after individuals handled the coral without protective gear. Multiple aquarium store employees, along with all the residents of a home who were exposed to the coral, developed symptoms that mostly resolved by the following morning.

There are various other case reports of palytoxin exposure. One case was that of a 25-year-old female who handled a zoanthid coral from her home aquarium and subsequently developed perioral paresthesia and dysgeusia (alteration of the sense of taste) that lasted for several days. She was treated with corticosteroids and antihistamines.3 Another case involved a patient who experienced dizziness, generalized weakness, myalgias, electrocardiogram abnormalities, and rhabdomyolysis. The symptoms resolved after supportive treatment with intravenous fluid.4

Though the neurology team suspected HPP in our patient, it is unlikely that this would have a first-time onset at 34 years of age. HPP begins in the teenage years and may manifest as several attacks per week. It may be precipitated by rest after exercise, stress, or a high-carbohydrate meal. Our patient was healthy and exercised regularly. It was unlikely for him to suddenly develop HPP if there were no changes to his routine. The only change was the exposure to the coral.

Conclusion

It is easy in a busy emergency department to attribute a patient's symptoms to more common diagnoses. However, it is important to develop a broad differential and to obtain a detailed history. In our case, asking about the patient's pets revealed his exposure to zoanthid coral. There are no current guidelines on proper handling to avoid exposure to palytoxin. Warning signs may help with educating the general public about the corals. However, proper handling techniques need to be established to prevent future exposure. O

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BENZOS & CHILL

Treatment of toxin-induced hyperthermic disorders

BY DAVID TRAFICANTE, DO, AND JOHN KASHANI, DO

Case 1

A 39-year-old female is transferred to your emergency department from an outpatient same-day surgery center. The patient reportedly developed tachycardia and tachypnea while in the postoperative suite approximately 30 minutes after undergoing general anesthesia for a colonoscopy. She was sent to your emergency department for evaluation, where she was found to be hyperthermic with a rectal temperature of 106.5°F. You notice a rising end-tidal CO_2 despite no change in ventilation and severe masseter muscle spasm.

Case 2

A 73-year-old male with a past medical history of Parkinson's disease presents to the emergency department from home with fever and altered mental status. His vital signs are blood pressure 98/59, pulse 102, respiration 15, and temperature 102.6°F. On exam, you recognize "lead pipe" rigidity and bradykinesia. A septic workup is performed, but there is no obvious source of infection. His wife reports he stopped taking his "Parkinson's medication" a few days ago.

Case 3

A 27-year-old male with a past medical history of depression, currently treated with fluoxetine, is brought into the emergency department by his girlfriend for altered mental status. He is febrile with a temperature of 102.2°F, but the rest of his vital signs are within normal limits. He is agitated, is diaphoretic, and has lower extremity clonus. His girlfriend shows you a bottle of dextromethorphan and reports the patient started taking it yesterday for a cough.

The cases above describe three different hyperthermic disorders: malignant hyperthermia, neuroleptic malignant syndrome (NMS), and serotonin syndrome. These syndromes are often confused with one another when being considered them in the diagnosis of a hot and stiff patient presenting to the emergency department (see Table 1 on pg. 8).

Malignant Hyperthermia

Malignant hyperthermia is a hypermetabolic syndrome of skeletal muscle, which can occur in individuals with inherited defects of their ryanodine L-type calcium channels. These calcium-release channels are located in the sarcoplasmic reticulum of skeletal muscle and dump out large quantities of calcium ions when stimulated by exposure to certain agents.1 The increase in intracellular calcium levels in skeletal muscle causes increased muscle contraction and increased consumption of adenosine triphosphate, releasing excessive heat, resulting in hyperthermia. The most common agents responsible for causing malignant hyperthermia are certain volatile gas anesthetics (halothane, sevoflurane, desflurane) and succinylcholine. There have also been case reports of



excessive exercise and heat leading to the condition.²

The onset of symptoms is relatively rapid, typically presenting within minutes to hours. Diagnosis can occasionally be delayed until the postoperative period in patients undergoing general anesthesia, as described in the patient in Case 1. The most common presenting symptom is unexplained hypercarbia (a rising end-tidal CO, despite no change in ventilation), followed by hyperthermia, sinus tachycardia, and masseter muscle spasm.3 Often, these patients will develop rhabdomyolysis, which can lead to hyperkalemia, worsening acidosis, and even cerebral edema. The management of patients with presumed malignant hyperthermia begins with benzodiazepines for muscle relaxation, aggressive cooling measures, and early correction of hyperkalemia. The reversal agent for malignant hyperthermia is dantrolene (initial dose is 2.5 mg/kg IV), a hydantoin derivative that functions by inhibiting calcium release from the sarcoplasmic reticulum by antagonizing ryanodine receptors. Dantrolene can cause diaphragmatic weakness. Close monitoring of respiratory status is recommended. For patients who are untreated, mortality has been reported to be as high as 70 percent versus about 7 percent for those who receive proper management.⁴ The hotline run by the Malignant Hyperthermia Association of the

United States (800-644-9737) can be consulted for assistance in treatment.

Neuroleptic Malignant Syndrome

Case 2 describes the presentation of NMS, an idiosyncratic reaction secondary to an overall dopamine deficit caused by either excessive dopamine antagonism from certain antipsychotic agents (eg, haloperidol, olanzapine, quetiapine, risperidone) or

"Those individuals who were taking an antipsychotic medication as a cause for their NMS can be treated with bromocriptine, a dopamine agonist used to overcome dopaminergic blockade. There are case reports for the use of dantrolene in NMS. However, this remains controversial." from withdrawal of dopamine agonists (eg, Parkinson's patients who abruptly stop their dopamine agonist medications).⁵ The dopamine deficit in the hypothalamus and basal ganglia leads to hyperthermia as a result of extrapyramidal side effects like sustained muscle contraction and an elevated temperature set point. There is also a theorized removal of tonic inhibition from the sympathetic nervous system, which results in characteristic autonomic instability.⁶

The onset of symptoms for NMS is longer than the other hyperthermic disorders, typically presenting within days to weeks of starting or increasing the dose of a dopamine antagonist or withholding a dopamine agonist. The classic finding on physical exam is a catatonic patient with lead pipe rigidity and bradykinesia. Other symptoms include Parkinsonian-like manifestations including dystonia, akathisias, and resting tremors. Management again begins in the emergency department with active cooling and benzodiazepines for muscle relaxation. Patients with NMS are also susceptible to developing rhabdomyolysis, although it is less common than in patients with malignant hyperthermia.7 Those individuals who were taking an antipsychotic medication as a cause for their NMS can be treated with bromocriptine, a dopamine agonist used to overcome dopaminergic blockade. There are case reports for the use of dantrolene in NMS. However, this remains controversial. Parkinson's disease patients who present with NMS secondary to withdrawal should be restarted on their appropriate dopamine agonist medications. In addition, aggressive IV fluid hydration can help prevent acute renal failure and enhance excretion of muscle breakdown products. Each year, approximately 2,000 patients are diagnosed with NMS in the United States, with an overall mortality rate of approximately 10 percent.8 Disposition of patients with NMS, as with the other hyperthermic disorders, involves close monitoring in an intensive care unit.

Serotonin Syndrome

Case 3 represents a patient with serotonin syndrome. Serotonin syndrome presents as a constellation of cognitive, neuromuscular, and autonomic symptoms secondary to an excess of synaptic serotonin. The mechanism of this syndrome is quite complex and involves a combination of interactions involving central catecholamine release, the hypothalamic-pituitary-adrenal axis, the sympathetic nervous system, and skeletal muscle. Often, as described in this case, the syndrome is seen in patients who ingest medications that synergistically increase serotonin, although it can also occur from an overdose of a single serotonergic agent.⁹ Some of these drugs are listed in Table 2, see p. 8.

The symptoms of serotonin syndrome are **CONTINUED** on page 8



What emergency physicians need to know about complex regional pain syndrome

BY JONATHAN GLAUSER, MD, **MBA, FACEP, AND SARAH** MONEY, MD

reviously called reflex sympathetic dystrophy (RSD) or causalgia, complex regional pain syndrome (CRPS) entails severe and chronic pain and disability involving a

part or whole of a limb. The pain follows a non-dermatomal distribution. CRPS-1 (reflex sympathetic dystrophy) is the most common, accounting for approximately 90 percent of cases, and differs from CRPS-2 (causalgia) in that a nerve lesion typically from trauma, vascular event, or surgery is identified in the latter. Approximately 60 percent of cases involve the upper limbs, with 40 percent affecting a lower limb. CRPS typically follows a minor or moderate extremity injury, such as a wrist fracture, sprain, blunt injury, stabbing, animal bite, or elective surgery. In a minority of cases, no inciting event can be recalled or identified. The disorder typically starts within four to six weeks of the injury. Most patients with CRPS report that pain caused them to stop working.1 It may follow a stroke or myocardial infarction ("shoulderhand syndrome"). The incidence has been reported to be as high as 28 percent following Colles' fracture, although most cases resolved after one year. Approximately 3 to 5 percent of patients who sustain a distal radius fracture develop CRPS.²⁻⁴ Muscle weakness and changes in sweating and hair and nail growth may occur in that limb.⁴ Allodynia is defined as pain induced by non-painful stim-



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This condition is also known as reflex sympathetic dystrophy. The symptoms can worsen over time and spread through the body as they are reinforced in the cycle shown here. The sympathetic response (green) from the brain (upper right) to the pain impulses (blue) cause vessel spasms (lower right) that increase the pain. In this case, the pain and swelling are in the hand, but other areas of the body can also be affected. It is severely painful and treatment is difficult, involving drugs, electrical stimulation, and psychological and neurological forms of therapy.

uli (painful touch), whereas hyperalgesia is more pain than expected to be induced by painful stimuli. One or both should be present in making this diagnosis. Continuing pain in CRPS is disproportionate to the inciting event. Pain, sensory, trophic (hair growth increase, changes in nail growth), and motor symptoms are not confined to single nerve innervation territories.3 Vasomotor findings may include temperature asymmetry, skin color changes, sweating changes/asymmetry, or edema.5

There may be objective findings in the patient with CRPS-for example, side-to-side difference in skin temperature, or osteopenia and osteoporosis with abnormalities on bone scan/scintigraphy or on plain X-rays with both hands on one film. Other potential testing may include MRI, which may demonstrate bone marrow edema and autonomic testing of sweat output. The diagnosis of CRPS is excluded by the existence of another condition that would better explain the degree of pain and dysfunction.⁶ Women are affected more often than men. It is most frequent in those ages 61 to 70.7 Approximately 20 percent of patients are able to resume previous activities.8

The diagnosis of CRPS has historically been one of exclusion. The Budapest criteria were established in 2003 to objectively diagnose CRPS and have been shown to have a sensitivity of 99 percent and a specificity of

CONTINUED on page 8



79 percent.⁶ These four criteria must be met for the diagnosis: 1) pain out of proportion to the inciting injury; 2) patient's reported history of at least one symptom in three of the four categories of sensory (eg, hyperesthesia, or increased pain response to a mildly painful stimulus, and allodynia, or a painful response to a nonpainful stimuli), vasomotor changes (eg, temperature or skin color asymmetry), sudomotor/edema changes (eg, asymmetric limb edema or sweating), and motor/trophic changes (eg, decreased range of motion; weakness or atrophy; tremor; or hair, nail, or skin changes); 3) the clinician's own observation of at least two symptoms that fall into two of the previously mentioned categories; and 4) no other explanation for the patient's symptoms (see Table 1).

Treatment

Several treatment options are available. There is evidence for posttraumatic inflammation in CRPS, so nonsteroidal anti-inflammatory drug or steroid therapy in the acute stage is reasonable. A high dose of prednisolone has been proposed as therapy.⁶ Additional oral medications that can be used include tricyclic antidepressants and anticonvulsants such as gabapentin and carbamazepine. There is some evi-

Table 1. Budapest Clinical Diagnostic Criteria for CRPS⁶

1. Continuing pain that is disproportionate to any inciting event.

2. Must report at least one symptom in three of the four categories:

- a. Sensory: hyperesthesia and/or allodynia
- b. Vasomotor: temperature asymmetry and/or skin color changes and/or skin color asymmetry
- c. Sudomotor/edema: reports of edema or sweating changes and/or sweating asymmetry
- d. Motor/trophic: decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. Must display at least one sign in two or more of the following categories:

- a. Sensory: hyperalgesia to pinprick, allodynia to light touch and/or deep somatic pressure and/or joint movement
- b. Vasomotor: evidence of temperature asymmetry and/or skin color changes and/or asymmetry
- c. Sudomotor/edema: evidence of edema and/or sweating changes an/or sweating asymmetry
- d. Motor/trophic: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. No other diagnosis that better explains the signs and symptoms.

dence that gabapentin or carbamazepine therapy may reduce pain in CRPS.⁶ Bisphosphonates inhibit the activity of osteoclasts. Pamidronate 60 mg as one intravenous dose has been proposed.9,10

Topical dimethyl sulfoxide (DMSO 50 percent cream) has been shown to have a positive effect on pain, presumably through its role as a free-radical scavenger. Applied for two months, it may provide significant pain relief.¹⁰ Other topical medications such as capsaicin, lidocaine 5 percent, or eutectic mixture of local anesthetics (EMLA) cream may be used as well. Patients should be encouraged to use the affected extremity. Specifically, splints, slings, and immobilizing devices, particularly for prolonged periods, should be avoided.9

Intravenous immune globulin has been used effectively, with the rationale that CRPS is related to the presence of unidentified neu-

BENZOS & CHILL | CONTINUED FROM PAGE 6

extremely variable, ranging from mild presentations to a life-threatening syndrome. The most common presenting symptoms are muscle rigidity, altered mental status, and hyperthermia. Classically, and differentiating this condition from the other hyperthermic disorders, patients develop myoclonus, which is characteristically more evident in the lower extremities. The onset of symptoms is typically rapid, with symptoms typically presenting within minutes to hours. The cornerstone to management of serotonin syndrome involves immediate discontinuation of all serotonergic agents as well as supportive treatment with aggressive cooling and benzodiazepines. Current recommendations include the use of cyproheptadine, an antihistamine with antiserotonergic properties, and chlorpromazine, a phenothiazine antipsychotic with antiserotonergic properties.¹⁰ Mortality rates are similar to that of NMS, between 2 and 12 percent.

Treatment

Management of patients with the described toxin-induced hyperthermic disorders begins with supportive care and focuses on decreasing muscle activity (benzos) and core body temperature (and chill). Benzodiazepines play an important role in decreasing mortality by reducing shivering and muscle breakdown that can lead to rhabdomyolysis, hyperkalemia, and ultimately renal failure. Patients with severe toxicity who do not respond to benzodiazepines may even require chemical paralysis with a non-depolarizing paralytic agent along with mechanical ventilation for better control of their muscle hyperactivity. •

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Table 1. Management of Toxin-Induced Hyperthermic Disorders

	MALIGNANT HYPERTHERMIA	NEUROLEPTIC MALIGNANT SYNDROME	SEROTONIN SYNDROME
INCITING AGENT Pharmacology	CITING AGENT ARMACOLOGY Inhalational anesthetics, succinylcholine Neuroleptic antipsychotic agents (dopamine antagonists) or withdrawal of Parkinson's disease medications (dopamine agonists)		Serotonergic agents and drugs of abuse
ONSET OF Symptoms After Exposure	SET OF MPTOMS AFTER Minutes to hours Days to weeks OSURE		Hours
MENTAL STATUS	Agitation	Catatonic, mute	Confused, agitated
MUSCLE Symptoms	Bradykinesia	Bradykinesia Catatonic, lead pipe rigidity, hyporeflexia	
VITALS	Severe hyperthermia, autonomic instability	Mild hyperthermia (<102.5° F) Autonomic instability	
TREATMENT	Initial support: cooling, muscle relaxation	Initial support: cooling, muscle relaxation	Initial support: cooling, muscle relaxation
	Reversal agent: dantrolene	Reversal agent: bromocriptine	Reversal agent: cyproheptadine, chlor- promazine

Table 2: Drugs That May Cause Serotonin Syndrome DRUGS

Antidepressan	ts
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CLASS

Antidepressants	s Selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, serotonin and norepinephrine reuptake inhibitors, tricyclic antidepressants, buproprion, trazodone Fentanyl, tramadol	
Analgesics		
Cold/cough preparations	irations Oral decongestants, dextromethorphan	
Psychedelics, illicit drugs	MDMA, amphetamines, cocaine, LSD	
Antibiotics	Linezolid	
Herbs	St. John's wort, ginseng, nutmeg, yohimbe	
Others	Lithium, metoclopramide, ondansetron, triptans, levodopa, valproate	

ral antibodies. However, use in the emergency setting has not been recommended.¹¹ The role of opioids as a second- or third-line option for therapy ("rescue dosing") has not been completely defined. Opioids have not been recommended, except with specific advice and input from a pain management specialist.⁹ There are concerns for tolerance, cognitive impairment, and opioid-induced hyperalgesia.⁶ Non-pharmacologic treatment options include appropriate physical and occupational therapy regimens such as isotonic strengthening, passive gentle range of motion, aerobic conditioning, aquatic therapy, and ergonomics.

Vitamin C is proposed to prevent CRPS. However, there is not enough evidence to recommend this for routine therapy following, for example, a Colles' fracture. More invasive therapies exist but are outside the scope of emergency medicine. Spinal cord stimulation and intrathecal baclofen pumps have led to a decline in pain. Stellate ganglion block, brachial plexus block, and lumbar sympathetic block have all been used with some success.⁶

The role of lidocaine or ketamine in the management of chronic painful conditions is evolving. Topical lidocaine comes in patches, creams, and ointments. These have been used in CRPS. Options for CRPS include EMLA and 5 percent lidocaine-impregnated patch.¹² Intravenous lidocaine has been evaluated in patients with renal colic and for acute low back pain. It has been proven effective in resolution of pain when used in doses ranging from 1.5 mg/kg to 100 mg IV. Side effects of nausea and dizziness tend to be mild and transient. One study evaluated the use of IV lidocaine infusion in CRPS patients. There was evidence for a decrease in pain response to cold stimuli and, at 3 mcg/ mL infusion, a decrease in pain level.¹³ Lidocaine infusions have been advocated for severe and neuropathic pain, as in cancer or diabetic neuropathy. Dosage for testing purposes has been proposed at 1-3 mg/kg (100 mg being a frequently used dose) over 20 to 30 minutes. If the challenge dose is effective, then an infusion at 0.2–2 mg/kg per hour can be given, often with dramatic relief of pain.¹⁴ At this time, the role of intravenous lidocaine in the management of neuropathic pain remains to be defined.

Ketamine has been demonstrated to be efficacious in the management of pain in CRPS.¹⁵ It prevents or attenuates the hyperalgesia and allodynia of CRPS.¹⁶ As noted below, it may prove to be effective in inducing long-term pain relief when administered for four to 14 days.¹⁷

Intravenous ketamine in subdissociative doses may be effective in CRPS. A recent study of low-dose ketamine when used as an infusion initially of approximately 5 mg/hour titrated up to as much as 30 mg/hour for a 70 kg patient over 4.2 days in 60 patients with CRPS demonstrated significant pain relief during a 12-week study period, not simply during its administration.¹⁸ The chance for clinically significant pain reduction for 11 weeks after therapy must be weighed against the cost of hospitalization.

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Reviewing ILCOR Guidelines on Asthma, Concussion, Dental Avulsion, and Anaphylaxis

Dissecting the evidence behind the recommendations

Editor's Note: This is part two of a four-part series.

International Liaison Committee on Resuscitation he International Liaison Committee on Resuscitation (ILCOR) appointed a task force in 2013 to prepare recommendations regarding first-aid care by trained or untrained rescuers. The recommendations were released with the 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. The goal was to provide an evidence base for the initial care provided by laypersons, EMS, and physicians outside of the office or hospital setting.

ACEP Now has partnered with three emergency medicine residency training programs (Wake Forest School of Medicine, Winston-Salem, North Carolina; Mayo School of Graduate Medical Education/Mayo Clinic, Rochester, Minnesota; and Warren Alpert Medical School of Brown University, Providence, Rhode Island) to review 15 of these recommendations following the PICO (Population, Intervention, Comparator, and Outcomes) analytic format utilized by the recommendation authors.

Panel Commentators

Howard Mell, MD, MPH, CPE, FACEP, assistant professor, Wake Forest Baptist Medical Center, Department of Emergency Medicine
Jessica L. Smith, MD, FACEP, associate professor (clinical), Warren Alpert Medical School of Brown University, and program director, Emergency Medicine Residency

• Jason Stopyra, MD, FACEP, assistant professor, Wake Forest Baptist Medical Center, Department of Emergency Medicine

• Matthew Sztajnkrycer, MD, PHD, FACEP, associate professor, Mayo Clinic, Department of Emergency Medicine

Reference: Singletary EM, Charlton NP, Epstein JL, et al. Part 15: first aid: 2015 American Heart Association and American Red Cross guidelines update for first aid. *Circulation*. 2015;132(suppl 2):S574–S589.

BRONCHODILATORS FOR ASTHMA WITH SHORTNESS OF BREATH (FA-534) Recommendation Author: Bradley M. Chapman, MD

Dr. Chapman is a member of the emergency medicine residency training class of 2018 at the Wake Forest School of Medicine. **QUESTION:** Among adults and children in the prehospital setting who suffer from asthma and are experiencing difficulty in breathing (P), does bronchodilator administration (I) compared with no bronchodilator administration (C) change time to resolution of symptoms, time to resumption of usual activity, complications, harm to patient, therapeutic endpoints, and need for advanced medical care (O)?

Results: Eight double-blind randomized controlled trials (RCTs), two observational studies, and one meta-analysis were identified that addressed the PICO but were all deemed very-low-quality evidence.

Outcomes: Regarding the critical outcome of time to resolution of symptoms, two RCTs showed benefit in reduction of time to subjective improvement in dyspnea in participants treated with fast-acting bronchodilators. Regarding complications, two RCTs and one observational study demonstrated little or no difference between participants treated with bronchodilators and those treated with placebo. Regarding the therapeutic endpoints of oxygenation and ventilation, two RCTs showed benefit in an improvement in forced expiratory volume over one second when comparing inhalers to placebo.

Discussion: This review found evidence that use of a bronchodilator in asthmatics with acute difficulty breathing is effective for reducing wheezing, dyspnea, and respiratory rate while improving measures of effectiveness with few reported side effects.

Recommendation: When an individual with asthma is experiencing difficulty breathing, trained first-aid providers should assist the individual with administration of a broncho-dilator.

Note from Dr. Stopyra: Inhaled bronchodilators are beneficial to patients and have very low risk of complication. If they are available outside of the hospital, they should be used.

CONCUSSION (FA799) Recommendation Author: Heather Rybasack-Smith, MD

Dr. Rybasack-Smith is a member of the emergency medicine residency training class of 2017 the Warren Alpert Medical School of Brown University. **QUESTION:** Among adults and children with concussion defined as suspected head injury (P), does use of a scoring system (I) compared to standard first-aid assessment (C) change outcomes (O)?

Results: One retrospective observational study was identified that addressed the PICO and was deemed appropriate for first-aid providers. The study rescored prehospital Glasgow coma score (GCS) from trauma registry data with a simple motor score. Scores were compared to four outcomes. This study was deemed low-quality evidence.

Outcomes: For differentiation between minor and severe concussion and need for advanced care, no significant difference was observed between the motor score versus the GCS. For change in time to recognize clinical deterioration, time to transport, risk of poor neurologic outcome, and 30-day survival, no evidence was identified.

Discussion: Recognition of concussion by first-aid providers is difficult. Signs and symptoms of concussion are complex and may be subtle and progressive. Failure to recognize a concussion and refer for further evaluation, or failure to remove from sports activities, can result in significant morbidity and mortality. The use of a simple validated tool to identify concussions is valuable but would need robust evidence to support its use by first-aid providers. The task force identified several tools that used before-andafter assessments, but these were felt to be impractical for standard first-aid use. The task force found no evidence to support or refute the use of any simplified tool to identify significant concussion.

Recommendation: No recommendation.

Note from Dr. Smith: Individuals with head injury and any evidence of alteration of consciousness require evaluation by advanced health care providers or evaluation in an emergency department.

DENTAL AVULSION (FA 794) Recommendation Author: Andrew Escamilla, MD

Dr. Escamilla is a member of the emergency medicine residency training class of 2017 at the Mayo Clinic School of Graduate Medical Education/Mayo Clinic. **QUESTION:** Among adults and children with an avulsed permanent tooth (P), does storage of the tooth in any solution prior to replantation (I) compared with storage in whole milk or the patient's saliva (C) change success of replantation, tooth survival or viability, infection rate, pain, malfunction (eg, eating, speech), or color of the tooth (O)?

Results: Eight studies were identified comparing the success of replantation and survival in Hank's balanced salt solution (4), propolis (2), egg white (2), coconut water (1), Ricetral (1), saline (2), or phosphate-buffered saline (1) compared with whole milk. Based upon the available data, early immersion in Hank's balanced salt solution, propolis, egg white, coconut water, Ricetral, whole milk, saline, or phosphate-buffered saline is beneficial (in order of preference). There is insufficient evidence for or against temporary storage of an avulsed tooth in saliva compared with alternative solutions. There was no evidence to address the important outcomes of infection rate, pain, malfunction, and cosmetic outcome.

Discussion: An avulsed tooth constitutes a dental emergency and can result in permanent loss of the tooth. Tooth survival is dependent on early replantation. Previous recommendations state that the tooth should be placed in a variety of storage media in situations that do not allow for immediate replantation. Based upon current data, rapid assistance with re"Nine studies showed benefit from repeat administration of epinephrine, while a single study demonstrated no difference in resolution of symptoms when comparing one dose versus two doses. None of these studies specifically addressed adverse effects or complications from multiple epinephrine doses."

plantation should be sought. The use of a particular storage medium should depend upon availability and should not delay efforts at replantation.

Recommendation: If a tooth cannot be immediately replanted, the preferred storage medium is Hank's balanced storage solution (weak recommendation, very-low-quality evidence). Whole milk is preferred to saline solution as a temporary storage solution (weak recommendation, very-low-quality evidence). Note from Dr. Sztajnkrycer: The dental guidelines assume an ideal world in which Hank's balanced salt solution is available to first-aid providers, especially in a remote setting in which immediate replantation is not a viable option. While there was insufficient evidence supporting the use of saliva as a temporary medium, there was also insufficient evidence against this. Whole milk is also an option.

ANAPHYLAXIS (FA 500)

Recommendation Author: Eric C. Funk, MD *Dr. Funk is a member of the emergency medi*-

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before March 24, 2017 acep.org/pemassembly cine residency training class of 2017 at the Mayo Clinic School of Graduate Medical Education/Mayo Clinic.

QUESTION: In patients with anaphylaxis who do not experience resolution of symptoms after receiving the first dose of epinephrine (P), does administering a second dose of epinephrine (I) compared to not administering a second dose of epinephrine (C) change outcomes (O)?

Results: Ten observational studies were identified, all of which were categorized as very low quality due to risk of bias and confounding variables.

Outcomes: Nine studies showed benefit from repeat administration of epinephrine, while a single study demonstrated no difference in resolution of symptoms when comparing one dose versus two doses. None of these studies specifically addressed adverse effects or complications from multiple epinephrine doses. **Discussion:** Despite limited data, the benefit

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of resolution of life-threatening symptoms, including compromised airway, difficulty breathing, and hemodynamic collapse, appears to outweigh the potential risks. While a time frame for administration of a second dose was not addressed by the current literature review, previous studies suggest 10 to 15 minutes between the two doses. There are reports in the literature of adverse effects following incorrect doses or routes of administration. Auto-injectors may reduce the occurrence of these adverse events.

Recommendation: A second dose of epinephrine should be administered to patients who are experiencing severe anaphylaxis and who have not had a resolution of symptoms after the initial dose (weak recommendation, very-low-quality evidence).

Note from Dr. Mell: While the data only weakly support the practice in the prehospital environment, this is the standard practice in emergency medicine. There's no reason to withhold a second epinephrine dose (if needed) outside of the hospital.



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Discharge Early to Improve Hospital Capacity and Flow

A team approach was essential to meeting NYU's goal of earlier discharge any hospitals in the country operate at capacity, and many patients are boarded in the emergency department. Although there are many ED-based flow initiatives, virtually none of these address the most significant impediment to flow: boarding of admitted patients in the emergency department due to lack of inpatient beds. Only a few interventions really have any lasting and significant impact on boarding and capacity.

This is one of a series of interviews that highlight dramatically effective interventions to reduce boarding and crowding. Early-morning discharges can have a strong impact on capacity, virtually eliminate boarding, and also decrease the length of stay. I recently sat down with Katherine Hochman, MD, to discuss NYU Langone Medical Center's efforts to increase capacity by discharging patients earlier in the day.

Participants

• Peter Viccellio, MD, FACEP, is vice chairman of the department of emergency medicine and associate chief medical officer for the Health Sciences Center at Stony Brook University in New York.

• Katherine Hochman, MD, is assistant professor and associate chair for quality of care in department of medicine at NYU Langone Medical Center in New York City.

PV: Welcome and thank you for joining me to discuss another issue related to hospital overcrowding and flow. As you know, this is a major issue for emergency medicine, hospitals, and patient safety. Previous literature has suggested that early discharge could really have a dramatic impact on hospital capacity and on hospital flow. You have been a leader in some major initiatives at NYU in order to improve early discharge. **KH:** Thanks very much for having me. The major metric that changed how we viewed early discharge was the fact that patients who came up to the floor after 1 p.m. stayed an average of 0.6 days longer even after you adjusted for all the different diagnoses compared with those patients who physically arrived on the floor before 1 p.m. This was a major eye-opener for us; the metric that really changed it all was that 0.6 increase in length of stay.

PV: There are five or six articles that looked at length of stay as a function of boarding, and the punchline was that there was roughly a day increase in stay if you boarded patients in the emergency department. When we initiated the full-capacity protocol, we reversed that. We were always curious as to why. If you admit and board a patient in the emergency department, they are going to get their antibiotics and their CT scans, but they're not going to get care management, social work, and other inpatient services. If you don't get them up in the morning, they lose that day.

KH: Discharge planning should be started on the day of arrival. You absolutely lose the efficiencies that you have with the team if a patient is in the emergency department and they don't have that team surrounding them. We started this discharge-before-noon initiative, and it absolutely was a multidisciplinary team effort. We knew that we were only as strong as the weakest link. If housekeeping didn't come up and clean the room, you could have a whole floor of rooms almost ready to go. We had a major kickoff event in which anybody who touched a patient or had anything to do with patient care was included. We jam-packed everybody into one of the conference rooms and discussed this before-noon initiative. We wanted to really hammer home the point that it's better patient care if the patient is able to safely leave the hospital-the earlier, the better. We don't want patients acquiring hospital-acquired illnesses, infections, or complications because they are hanging around waiting for lunch or for dinner. If a patient left before noon, the patient could get their medications from the pharmacy. If there was trouble with a pharmacy, someone would be around to field the phone call, and patients would be able to make their follow-up visits in the light of day. Not only was it important for the patient to be discharged, but never sacrificing safety meant that the patient in the emergency department could come up to the floor, the patients in the ICUs could come down to the floor, and also the post-anesthesia care units could become decompressed.

PV: Was this something that you started on medicine?

KH: There had been a goal set by the administration for a 30 percent discharge-before-noon rate. It hadn't been achieved for several quarters. I would say it was definitely a combined effort of administration and frontline staff and key medicine and nursing leadership.

PV: Usually when you get such a group together, there's a cacophony of voices explaining why you can't. How did you deal with all that?

KH: It certainly was an issue. Right before we did the kickoff, I had one of our administrative fellows at the time, Martha Bailey, come around with me while I was on the wards, and I asked Martha to write down the reasons why patients were not being discharged before noon. For 30 patients that we discharged, there might have been 40 different reasons why the patients did not go before noon. They included, "I wanted to stay for lunch," "I didn't know that I was being discharged," "I don't have a ride," "I don't have



"At 9 a.m., nurses and doctors, social workers, and

care managers all get together and talk about the issues for the day. One of these issues is early discharge. The team identified the three to six patients on their unit that are being discharged home, and the doctors have already rounded on them; those people could be discharged early."

-Katherine Hochman, MD

any clothing," "My family doesn't know." We realized very quickly that there wasn't a single answer that was going to fix this problem. That's why we involved every member of the interdisciplinary team, so that everyone knew the plan for the patient. We even had a way of prioritizing patients who needed a specific study or specific lab tests. Those patients would get prioritized first to get their study done so that we could make a decision on the day of discharge.

PV: To what degree was the physician workday impacted by this process? In many places, they're not finished with rounds until 11 a.m. or noon, and they don't do discharges until after that.

KH: We tried to work smarter instead of harder. It meant adjusting the resident conferences from the morning conferences to the afternoon. We also created a daily safety huddle in the morning. At 9 a.m., nurses and doctors, social workers, and care managers all get together and talk about the issues for the day. One of these issues is early discharge. The team identified the three to six patients on their unit that are being discharged home, and since the doctors have already rounded on them, those people could be discharged early.

PV: Before we get to the nitty-gritty details of the obstacles, what was the overall end result?

KH: The effort was started in March 2012. We quickly rose from the single-digit percentage of discharge before noon to over our target, which was 30 percent. We've sustained that over the years, and our most recent discharge-before-noon rate has been over 40 percent.

PV: What did it take to accomplish this?

KH: I think it is important to note that this effort did not cost the institution any money except for some pizzas, cupcakes, and a few gift cards. In terms of our process improvement, everything we did was one big, giant PDSA cycle [Plan Do Study Act]. On occasions when we missed a target discharge, we did a mini root-cause analysis with the medical director and nurse manager to determine why. Some issues were very actionable. Other times, we understood that, for example, a patient waiting for dialysis really should never have been put on the discharge-before-noon list, but we did work with hemodialysis staffing, and sometimes those nurses would come in early to accommodate a discharge-beforenoon patient.

PV: Did you have to extend more hours for care managers or social workers?

KH: No. Everybody, the care management and social workers, stayed during their current hours.

PV: What was the afternoon like before this process?

KH: Our interdisciplinary rounds were critical. Currently, between 1 p.m. and 2 p.m., the teams will round at the bedside. The hospitalist, the resident, the care manager, the social worker, and the nurse participate. We go around at the patient's bedside to answer four key questions. The first question is, why is this patient here? The second is, why is this patient still here? The third is, what has to happen for this patient to leave the hospital? The last question is, where and when will this patient be discharged safely?

PV: How self-sustaining is this? Does this still need to be prodded and pushed or has it become automatic now?

KH: It's definitely ingrained into our culture. When the care managers and social workers identify, along with the team, a discharge the day before, we put that in a computer program, and an email goes out to pretty much the entire medicine service and other services on a twice daily basis.

PV: Any pockets of opposition?

KH: The house staff had a small pocket. I think that we could have done a better job, perhaps, explaining the "why" to the house staff. The house staff constantly is rotating.

PV: Any steps going forward to further your progress?

KH: We're moving to a slightly different metric to capture our early discharges. We're gravitating to a median discharge time number. We've found that there's sort of a flurry of activity that occurs between 9:30 a.m. and noon where people are really trying to rally to get patients out, but we want to level the load a little bit. We want to avoid the dichotomy of 11:59 a.m. is a good discharge time, but 12:01 p.m. is a bad discharge time, so we're making a new metric called the median discharge time. The thrust of this new metric is, it's OK if you didn't make the 12:01 p.m. cutoff, but try and make every discharge as early as it can be. That's really the message.

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PASSING THE EM BATON | CONTINUED FROM PAGE 1





MODERATOR

Kevin M. Klauer, DO, EJD, FACEP, is an ACEP board member; chief medical officer–emergency medicine, chief risk officer, and executive director–patient safety organization at TeamHealth; *ACEP Now* medical Editor-in-Chief; and assistant clinical professor, Michigan State University College of Osteopathic Medicine, East Lansing.

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John Sherman, MD, FACEP, is an emergency physician at St. Elizabeth Healthcare; chair of the department of emergency medicine; and a charter member of ACEP.



FACEP, is president of Associated Emergency Physicians Medical Group APC in the greater San Diego area and ACEP President from 1985 to 1986.

KK: John, what was emergency medicine like when you and Harry started?

JS: Harry and I started at St. Elizabeth's July 1, 1971. We had no hospital status, no department, no representation on the executive committee, no leadership positions in the hospital, and, of course, we had no specialty status. We had to learn to make our own way. We look at things differently now. There's a lot more corporate medicine, and we have the benefit of clinical and even financial guidelines, regulations, and rules that ACEP has been instrumental in developing for us over the years. A lot has changed.

KK: John and Harry, what prompted you to start an emergency medicine group?

HM: John and I were on a ski trip while we were interns, and both of us had been working in emergency rooms as interns. Four of us in medical school decided to form a group. John and another were interning at St. Elizabeth's. I was interning up at Indiana University in South Bend. Another gentleman was in Minnesota. The possibility came about for St. Elizabeth's Hospital—who was using residents, family practice guys, and others to staff the emergency department—to form a group.

JS: I still have the original letter that I sent to the hospital administrator when Harry and I and two others had decided to form this group. This is a one-page proposal, \$15 an hour was what we asked for, and we offered to have a group of four physicians committed to St. Elizabeth's, committed to a career in emergency medicine. He signed it, and off we went.

ZJ: Today's residency graduates are being nearly inundated with the amount of job offers that we are receiving. When you guys were setting up your initial groups, was it more so out of necessity because there wasn't anybody offering the sort of position you were looking for or because you always had this entrepreneurial drive?

HM: Our hospital didn't have a need. They had been staffing the emergency department with different people, fly by night, and we came up with a proposal. After working in the emergency department as interns, we essentially ran the department. We had staff on call and another resident above us, but I enjoyed the "episodic-ness" of emergency medicine.

KK: Graduating residents are inundated with opportunities because there are far many more opportunities available than there are residency-trained, board-certified, or board-eligible emergency physicians. Back when you started your group in 1971, I'm sure you had to convince others who wanted to do other things with their life to pursue emergency medicine: "Come join us and be a part of this group while we are considering developing and evolving with this specialty."

RS: In 1971, when we first started here in San Diego, the idea of emergency medicine as a specialty was kind of unknown. It was just going to work in the pit! It was typically covered by anyone who could be coerced. Of course, there were no paramedics in those days. There weren't any trauma centers. We were the knife and gun club, and if you heard a honking horn outside, that meant there was a heroin overdose being dumped. We learned after a while, rather than extricate them at great personal risk, we'd just give them Narcan right in the back seat, and off they'd go. We delivered a lot of babies in the ED because women would wait in the parking lot until they were crowning. At that juncture, CAL/ ACEP was in its infancy. The importance of leadership in the early days became very clear. That's when EMPAC [the Emergency Medicine Political Action Committee] came along. Medicare was a problem. Around 1975, we marched on Washington with Terry Schmidt [our part-time lobbyist]. As our advocacy needs grew, along came NEMPAC. There were things we couldn't do in the ED. We couldn't intubate in the daytime because that was anaesthesia. We could do it at night because they weren't around.

KK: That was a great summary. Knowing that we are in a better place as a specialty, which era did you prefer to practice emergency medicine in?

RS: Well, that's not easy to answer, Kevin. It was certainly fly by the seat of your pants for sure. That part was exciting. I had no idea

of what I didn't know; none of us did. Going back to that perspective of the '70s, we would do things that would be unthinkable today. If someone was really nasty, we paralyzed them and talked to them, "You know, when I bag you, you breathe. When I don't bag you," you'd stop bagging for a while, "you notice you don't breathe. So I'm going to wake you up in a little while, and I want you to be really nice. Got it?" When they woke up, they were always nice.

Today, the rules are entirely different. The expertise we bring to the bedside today is much more exciting. Back then, we didn't have ultrasound, CT, or MRI. We had an IV, some blood, and that was about it.

KK: Zach, which world would you like to work in? In yesterday's emergency medicine or today's?

ZJ: Great question. I think it's comforting to hear people who've been around for a while saying that they're just as happy to practice today as they were initially. The entrepreneurial side of me thinks that it would have been advantageous to have been around 40 years ago. I think that starting your own group is challenging in today's environment. At the same time, I think a lot of the major battles have been fought, so it's certainly easier for folks to walk into emergency medicine today and be a respected member of the hospital and be respected as a specialist. I certainly thank everyone that came before us in terms of fighting all of those battles. In terms of where emergency medicine is headed in the future, I think that we've heard a lot more talk about being part of this acute care continuum and looking at how emergency medicine can get more involved in the prehospital environment, doing more things in terms of community paramedicine, keeping people out of emergency departments, how emergency physicians are more well-positioned to deal with unplanned episodic care, and looking at how new technologies are going to enable telemedicine to allow remote evaluation of patients.

JS: In the early '70s, a resident we helped train in the community told me that a leader, Don Thomas, told him that there were three things you needed to know in emergency medicine: Was the patient alive or dead? Are they going to live or die in the next 30 minutes? And did they need to be admitted, or could they go home? Like Richard was saying, there were times I did things I was never trained to do as a rotating intern, but it saved people's lives. Instead of having somebody to show you how to do something, you did it first, then you showed somebody else.

KK: I have a question for all three of you who've practiced much longer than Zach and I have. Was there ever a situation where you knew you were the only option for the patient?

RS: It was my first shift in 1971, and the moonlighting radiology resident couldn't go to work that night and said, "Could you go down there and work tonight?" I showed up, and they said, "Who are you?" I remember a patient walking in with an ice pick hole to the left of his sternum. He sat on the gurney; he was getting worse. I listened to his chest—Rice Krispies. I got an X-ray, and there was widening of his mediastinum, and I thought, "What the hell is this?" He promptly died on me. Had this guy come in a year later after I learned about opening chests, I probably could have at least performed a pericardiocentesis. It didn't take very long before I started doing things I really had no training in, and it was either, "You're going to die, or I'm going to try to do this."

JS: I had a young kid who was hit by a car, and most of the damage was to his face. He essentially had no airway. This kid needed an airway, so I had to do a cricothyrotomy on him, and I sent him off to Children's. The kid did great, but I was a nervous wreck for the rest of the shift.

HM: Well, you had to change your underwear fairly regularly when we were working in those days. That's improved.

JS: I think it really was more fun, but the patient care is much better now. There's just no question about it. We now have to put up with patient satisfaction surveys, be sure our charting is perfect, and follow all of the regulations. None of that was an issue back then.

was billing, insurance, or marketing, in order to make it all work. There was no plan. It was serendipity, but it turned out pretty well.

KK: So necessity bred invention. What about John and Harry? Did you feel that the support and development of ACEP helped you and your group?

JS: There was national ACEP, but California was a little ahead of Kentucky. We didn't even have a state chapter when Harry and I started. In fact, I got together with three other physicians around the state; we actually formed the Kentucky chapter of ACEP. National ACEP was huge because it gave us contact with other physicians who had common problems. There were meetings, and it provided a tremendous educational resource for us.

KK: Zach, from your perspective, what is the reputation of emergency medicine now among students who are deciding what specialty they want to practice?

ZJ: I think, reflecting on a couple of things that were said, that there have always been challenges in emergency medicine, and the

The entrepreneurial side of me thinks that it would have been advantageous to have been around 40 years ago. I think that starting your own group is challenging in today's environment. – Zachary Jarou, MD

After working in the emergency department as interns, we essentially ran the department. We had staff on call and another resident above us, but I enjoyed the "episodic-ness" of emergency medicine. – Harry F. Mills Jr., MD, ABEM

There were things we couldn't do in the ED. We couldn't intubate in the daytime because that was anaesthesia. We could do it at night because they weren't around.

- Richard Stennes, MD, MBA, FACEP

This kid needed an airway, so I had to do a cricothyrotomy on him, and I sent him off to Children's. The kid did great, but I was a nervous wreck for the rest of the shift. – John Sherman, MD, FACEP

RS: One of the questions you asked was about forming a group. In my case, it just sort of happened. My first partner was Nat Rose. We were in the Navy and got out in July 1972. We started working 16-hour nights for the first two years, rotating, Nat and I, and we were getting about \$20 an hour. We had an 80 percent contract to start. About four months into it, the hospital CEO said, "This doesn't work. We think we want to pay 60." That pushed us into independent billing. Inadequate Medi-Cal payments were a big issue. That made it clear that CAL/ACEP was a vehicle that we absolutely needed to have to make all of this work, and so three of us in my group, Bill O'Riordan, Roland Clark, and I, all became presidents of CAL/ACEP. We just evolved into things we needed to be and developed affiliate organizations, whether it challenges 40-plus years ago are a little bit different than the challenges that today's physicians are facing. It sounds like ACEP and other organizations are crucial in addressing these challenges, and I'm glad that those organizations are still around to help us today. I think a lot of people don't appreciate the history as much as everyone who was there and fighting these early battles. Nowadays, I think students probably take for granted that emergency medicine is a well-respected specialty with lots of resources within its own department at most every single major academic medical center and that emergency physicians are respected members of the health care team. Emergency medicine gets more and more applicants every year. It's very popular. I think that it seems on the same level as any medical specialty now, thanks to all the hard work from our pioneers.

JS: I get the sense from a lot of the younger doctors coming out of residency programs that ACEP is almost viewed as serving more the corporate or the business end of emergency medicine rather than the practicing physician. As a result of that, a lot of people don't even belong to ACEP. I've always felt being a member of ACEP was a good thing. Do you feel that some of that is being promulgated in the residency programs?

ZJ: As a resident with a busy clinical load, sometimes it's hard to appreciate the effect that ACEP has made in terms of lobbying to make sure that emergency physicians get fair reimbursement and are being treated properly within the health system. I think some residents may not understand the total impact that being part of ACEP has, and I think that one of the challenges is conveying that value to them as they transition out of residency, where their dues are often being paid for them, to demonstrate the value so they'll continue to pay when it comes out of their own pockets.

RS: Part of our problem with messaging to younger people is maybe we're not communicating what ACEP really does in terms of preventing things from happening in state capitals and preventing bad things from happening in Washington as well as making good things happen. Any one decision there can make a lifetime of difference for any one doctor and pay for their dues for a lifetime. Many may not know or understand what organized medicine, ACEP for the emergency medicine profession and the AMA in general, really does to make their lives possible, to protect their practice, and help them earn a living.

KK: Listening to Zach's passion, it's equal in magnitude but from a different perspective. Richard made a comment about the importance of "carrying the water" by advocating for and leading the specialty, and that resonates with me, and I'm sure it does with others, too. If the younger physicians out there are allowed to participate and carry some of that water, not as a burden but as an opportunity, then will they be more invested in the future of the specialty and with ACEP? We have to look for those opportunities. What would you want people to do so that what you've built doesn't erode and is protected?

HM: Richard speaks about carrying the water, but I think you have to chop the wood, too. I think we have to remember that we don't exist if there aren't patients. I had to always remember that they were the reason why we were there, to take care of people, and as long as you remember that the patients are the reason you're there, then all this other small stuff simply matters a whole lot less.

KK: The whole specialty owes a debt of gratitude and thanks to physicians like Harry, John, and Richard. If they had not accomplished what they had with yesterday's emergency medicine, Zach and today's emergency physicians would not be able to build a more perfect future for tomorrow's emergency medicine. •

Emergency physician leads US Olympic fencing team to victory

mergency medicine had a hand in this year's Olympic victories for the United States. Emergency physician **Ann Marsh-Senic**, **MD**, **FACEP**, served as captain of the fencing team in the 2016 Olympics. The team had a successful showing at the Rio games, bringing home silver medals in men's individual foil and men's individual sabre and bronze medals in men's team foil and women's team sabre.

An emergency physician with Independent Emergency Physicians, Dr. Marsh-Senic works at two hospitals, St. Joseph Mercy Oakland in Pontiac, Michigan, and Providence-Providence Park Hospital in Southfield, Michigan.

ACEP Now Medical Editor in Chief Kevin Klauer, DO, EJD, FACEP, recently sat down with Dr. Marsh-Senic to learn about her experiences leading the US fencing team and how she balances those responsibilities with her career as an emergency physician. Here are some highlights from that discussion.

KK: Tell me about your evolution. How did you transition into a leadership role?

AM: For fencing, there's really just one captain for the whole team. The fencing team is basically just one team. I guess you can say there are women's and men's, but we don't think of it as a separate team. I competed in 1992, 1996, and 2000, and my coach was the national coach back then. A couple of years ago, he actually asked me to start traveling with them to some World Cups to have someone to bounce ideas off of when he's in the box when the fencers are competing. He wanted my input on when he should substitute, different strategy ideas, and things to say to the fencers. My husband still competes in championships and World Cups sometimes. We actually went to the world championships in 2015 because my husband was competing. My husband is from a different country, so he competes for them.

KK: When did you last compete?

AM: Officially, I don't compete internationally anymore seriously—but I still compete in national events.

KK: Now I have to ask you, with fencing, it all looks pretty harmless, but did you ever inflict any bodily injury on anyone with the foil?

AM: You just get bruises and stuff. It's more likely to have an ankle or knee injury or something like that.

KK: You said you were helping advise the coach on when you were going to substitute. Do you tap out in fencing?

AM: We have a few events, and so for that particular event, it's a relay. But it's different than in other relays because you don't fence just the one person—you fence everyone on the other team. There are four people on the team, but there are actually three



people competing. Alternates can go in and

KK: When were you named the captain

AM: In December 2015. After the world

championships last year I wrote them a

letter saying I'd be interested in doing it for

the whole team. US fencing has like a sports

performance committee that nominated

me, and then the president appointed me

KK: Well, that is really exciting. Have

there been other female captains for

AM: Yes, I guess. I never thought about it.

KK: When you are thinking of gender eq-

uity issues, any time a woman aspires to

a new leadership role, I think its worthy

of pause and it's important to take no-

tice. I understand you are very humble,

and it probably didn't cross your mind

that you were breaking a glass ceiling of

any kind with this. But maybe you have.

AM: I never really thought of it. I've only

thought of it in the ways that I interact with

the coaches because all of the national

KK: So you were selected because you

KK: I'm very interested to hear about your

Olympic experience this past summer.

What was the venue like? I heard about

contaminated water and security issues.

AM: The honest truth? The venue was

great. Before every Olympics, the media

come back out.

of this team?

as the captain for 2016.

this team?

AM: Actually, no.

coaches are males.

AM: Yeah.

were the right person.

KK: That's very exciting.

Left: Daryl Homer (left), winner of the silver medal in the individual men's sabre at the Rio Olympics, with Ann Marsh-Senic. Bottom left: Men's foil team, who won bronze medals at the Rio Olympics. Left to right: Alexander Massialas, Gerek Meinhardt, Ann Marsh-Senic, Miles Chamley-Watson, and Race Imboden. Right: Alexander Massialas (right), winner of the silver medal in the individual men's foil at the Rio Olympics, with Ann Marsh-Senic.



goes off on these tangents about water quality or safety, or they aren't going to be ready in time. Really, we had a pretty seamless trip. The venue was spectacular, and I think the tournament was run really well. The main thing at the Olympics is to have on-time transportation that's reliable, and that was my only worry going into it, but the transportation was perfect.

KK: With your team, was there any particular challenge, injuries or illness, or some issue that made your role as captain really critical?

AM: All season, certain people on our team have had injuries that were always worrisome. However, we had a great medical person with us.

KK: Does it ever create any conflict at all when you probably outrank the medical person who is traveling with you?

AM: No because I don't want to be involved in that. Sometimes he'll ask me my opinion, but that's his specialty, sports injuries.

KK: How did your team do?

AM: We won four medals, which I think was a fantastic result. We got two silver medals and two bronze medals. I'm looking forward to Tokyo. I'm like, "Wow, we can win even more medals."

KK: After proving to yourself that you can, I think it makes it easier on yourself in the future. I have to ask a difficult question. Does the captain get a medal?

AM: No. The captains don't get a medal, just the athletes. What would I do with a medal?! Those are our medals though because that's our team. I'm sure that's how most people that are involved with the team feel.

KK: What do you see yourself doing in the future with fencing and/or emergency medicine? What are your aspirations?

AM: I'm happy to work my clinical shifts. But to be honest with you, I'm pretty likely to be doing the captain role until 2020. That's my focus right now for the next four years.

KK: How much of a time commitment does it take to be the captain of a team like this?

AM: This year, it actually took a lot of commitment because I became captain in the last year of the quadrennium. I had to travel with the teams I wasn't as familiar with. I went, during the eight months before the Olympics, to eight World Cups. They are mostly in Europe or in South America, so I have to take off four days from work or five days from work. Going forward, I have four years to spread it out over instead of eight months. I would think that just my main trips would be going to the world championships each year, which is about a week or a week and a half in the summer, and then potentially going to one or two World Cups a year, ramping it up a little bit more as it gets closer to Tokyo.

KK: What impact does that have on your personal life?

AM: I have two kids, so obviously I had to have more help in terms of watching them and taking care of them. But my husband was very supportive and thought I would be the right person for it, so he didn't complain too much. My kids are happy I'm not traveling right now. Actually, he told me that my presence might actually be more important than his, so I took that as a compliment.

KK: Eleven years of marriage, two kids, and a supportive spouse—that's absolutely great.

AM: Well, he's a fencer, too.

KK: Have you ever fenced each other?

AM: Yes, we have quite a few times. A lot of the events in the Midwest are mixed.

KK: Did you win?

AM: The first couple times, yes, but he hadn't fenced for 12 years. Now we try to avoid fencing each other because either he'll let me win and then I'm annoyed or he'll beat me and then I'm annoyed. My husband is a full-time coach, and so I help him run the fencing club here in my spare time. I actually went to Chicago last weekend to a competition and helped coach because he was also competing.

KK: Do your kids fence?

AM: They do. They just started in the last year or two. My kids are 6 and 9, so they are at the beginning of fencing. My daughter fences reluctantly.

KK: Well, maybe she'll be a future Olympic fencer—that's exciting.

AM: Yes, I think the bar has been set high. O

PEARLS FROM THE MEDICAL LITERATURE



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Concerns About SyncoPE

A heavy dose of spin on syncope and PE by RYAN PATRICK RADECKI, MD, MS

ave you thought about pulmonary embolism [PE]? I'll come see the patient, but would you mind putting in for a CTA? You know, almost a fifth of patients with syncope have PE."

Welcome to the hospital admission conversations of the future.

To great commotion and kerfuffle, the New England Journal of Medicine has delivered a new shiny pearl of medical evidence regarding the prevalence of PE in patients with syncope. The study in question, the Pulmonary Embolism in Syncope Italian Trial (PESIT), prospectively enrolled patients admitted from the emergency department with a first-time diagnosis of syncope.¹ These authors were concerned PE was an underappreciated cause of syncope in this population and undertook a systematic approach to evaluate its diagnosis. First, every patient admitted was risk-stratified using Wells criteria for PE. Then patients who were stratified as "PE unlikely" were tested using D-dimer. Finally, patients with positive D-dimer results and those who stratified as "PE likely" underwent imaging, either computed tomography pulmonary angiograms (CTPA) or ventilationperfusion lung scanning.

The shock and awe: 560 patients were included in the study, 230 underwent imaging or autopsy, and 97 received a diagnosis of PE. As summarized by the authors of the study: "In conclusion, among patients who were hospitalized for a first episode of syncope and who were not receiving anticoagulation therapy, pulmonary embolism was confirmed in 17.3 percent (approximately one of every six patients)."

This number is immediately eye-catching because it's entirely discordant with the 2 to 3 percent prevalence of PE in syncope from previously published literature.^{2,3} The authors contend their number is more accurate because of their systematic evaluation of consecutive first-time admissions regardless of associated symptoms or suspected underlying etiology. They conclude they have found these "occult" PEs as true missed causes of syncope. This is where your conversations with your admitting consultant may become increasingly frustrating. Chances are, they haven't delved into the details.

The first consideration is with regard to selection bias. These patients are higher-risk for PE than otherwise might be expected in the syncope population just based on their initial sorting. Of the 2,584 patients receiving a diagnosis of syncope in the emergency department, 1,867 were discharged home. We aren't given any information on these patients nor a description of specific testing performed in the emergency department other than a vague

In the syncope patient without any clinical symptoms relating to PE, it is reasonable to hypothesize some of these radiographic findings are unrelated and of uncertain clinical significance.

accounting of the suspected underlying benign etiology of syncope. However, in this greater context, the diagnosis of PE is made in 97 patients out of 2,584, or 3.8 percent of cases, which is much closer to the familiar 2 to 3 percent range. The cohort these authors are systematically evaluating, just based on their initial clinical selection, is more likely to have a serious underlying etiology for syncope, including PE.

Another important factor grossly inflates the number of PEs identified in this inpatient cohort: the many patients whose PE should have been identified in the emergency department prior to study inclusion. There were 60 patients admitted to the hospital documented as having clear and obvious signs of deep vein thrombosis (DVT) in their lower extremities but for whom apparently no objective evaluation for PE was performed. Following the authors' protocol subsequent to admission, two-thirds of these patients proved to have PE. Then an additional 77 patients exhibited tachypnea on admission, and another 107 exhibited tachycardia. Similar numbers for other risk factors for PE, such as previous DVT or PE, immobility, recent surgery, or active cancer were noted. Between 30 and 60 percent of these patients with physiologic changes or risk factors for PE were ultimately diagnosed with PE. It is legitimate to question whether these patients received an adequate ED evaluation considering PE as a diagnosis, since many appear clinically obvious. Indeed, the authors report only 24 of their 97 patients with PE were absent clinical signs or symptoms, suggesting their prevalence estimate is inflated not by occult PE but simply by PE missed by the emergency department.

Furthermore, the elephant in everyone's room is the troubling prevalence of false-posi-



tive imaging for PE, particularly when the pretest likelihood is low.⁴ In this cohort, 24 of 72 patients undergoing CTPA had found PE in segmental or subsegmental locations. Between one-quarter and one-half of PEs in these distal branches were found to be false-positives on subsequent subspecialty radiologist review.⁵ Another 24 patients received diagnosis of PE based on ventilation-perfusion scanning, and half of those found perfusion defects in between 1 and 25 percent of the area of both lungs. Again, as the size of the defect decreases, the likelihood of false-positives increases.

Lastly, we enter into the complicated question of whether these PEs are acutely related to the syncopal episode or whether they represent chronic or incidental findings. In the syncope patient without any clinical symptoms relating to PE, it is reasonable to hypothesize some of these radiographic findings are unrelated and of uncertain clinical significance. Attributing temporary global cerebral hypoperfusion to PE requires a complicated cascade of vascular obstruction, vasoconstriction, and right ventricular afterload. In the instance that these are incidental or represent overdiagnosis, it is reasonable to be concerned about the bleeding risks of long-term anticoagulation in this mostly elderly cohort. PE is not a zeromiss diagnosis for this precise reasoning. The risks of treatment outweigh the benefits for some patients diagnosed with PE.

At the end of the day, the important takeaway is this: These data don't generalize to our typical emergency department, which pursues a reasonable explanation for vital sign abnormalities and clinical signs suspicious for DVT. It is important to consider the diagnosis of PE in patients with syncope, but most of the patients with clinically important or true-positive PE will have obvious signs pointing to a need for objective testing. There is no indication this study reflects an otherwise unexpected population of PE in syncope following admission to the hospital. I fear this study will lead to an increase in harmful lowvalue overtesting.

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PRACTICAL TIPS FOR THE PRACTICAL DOC









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End-Tidal Capnography Is Not Just a One-Trick Pony: Part 2

Use capnography for placing orogastric/nasogastric tubes and CPR

by KATRINA D'AMORE, DO, MPH, TERRANCE MCGOVERN, DO, MPH, AND JUSTIN MCNAMEE, DO

Figure 1. Placement of a Side-Stream Capnometer (left) and Mainstream Capnometer (right)

n the last "Tricks of the Trade" column (November 2016), we reviewed how to use end-tidal capnography to detect diabetic ketoacidosis and monitor chronic obstructive pulmonary disease exacerbations. Now we'll explore using end-tidal capnography to check orogastric/nasogastric tube placement and guide cardiopulmonary resuscitation.

Using End-Tidal to Check Orogastric/ Nasogastric Tube Placement

Press Ganey scores likely skyrocketed once we stopped shoving nasogastric tubes (NGT) into every patient with the slightest evidence of an upper gastrointestinal bleed, but we are still placing orogastric (OGT) or NGT in our intubated patients in the emergency department. While insertion of these tubes is a relatively straightforward procedure, it can result in fatal consequences if there is inadvertent airway placement of the tube. This dire misplacement is even possible in intubated patients with an inflated endotracheal cuff.^{1,2} Typically, we check for aspiration of stomach contents and auscultate to confirm placement, but these can be unreliable indicators, especially in a busy emergency department.3 The gold standard for tube placement is visualization of the tube passing below the diaphragm on chest X-ray, which you could be waiting up to 87 minutes to receive.4,5

In theory, end-tidal capnograms are used to determine whether patients are hypoventilating, leading us to believe that using endtidal to check for unintentional pulmonary placement of NGT/OGT should be effective. With the increasing availability of end-tidal capnometry in the emergency department, it is convenient enough to temporarily attach one to the NGT/OGT (see Figure 1).

Meyer et al demonstrated that in 100 mechanically ventilated patients, a colorimetric capnometer was able to exclude tracheal placement 100 percent of the time.⁴ In this study, an NGT was initially inserted to a depth of 30 cm and insufflated and exsufflated with 50 cc of air, then the end of the tube was capped with the colorimetric capnometer. If the color remained purple, indicating an endtidal CO₂ (EtCO₂) <4 mmHg, the tube was advanced to an appropriate depth and secured. If the colorimeter changed to a yellow color, indicating EtCO₂>15 mmHg, the tube was removed. In a meta-analysis of nine clinical trials of mechanically ventilated patients, the use of either qualitative or quantitative capnometry for tracheal placement of the NGT had sensitivities ranging from 88 to 100 percent and positive likelihood ratios of 15.2-283.5.6 Before your patient leaves the emergency department, a chest

X-ray is needed to confirm placement of your NGT/OGT, but end-tidal capnography can help to avoid accidental airway insertion, multiple attempts at placement, and multiple chest Xrays. (See Figure 2 for an algorithm for placing NGT/OGT.)

End-Tidal to Guide Your Next CPR

A cardiopulmonary resuscitation (CPR) is inherently a scene of chaos with many unknowns, but arguably one of most informative pieces of information, aside from presence of a pulse, is the end-tidal capnogram. End-tidal capnometry made its debut in the advanced cardiovascular life support (ACLS) guidelines in 2010, but its use in cardiac arrest can be found as far back as the 1970s.^{7,8} It can be used for prognostication, quality of chest compressions, correct placement of the endotracheal tube, and perhaps even the cause of the arrest.

When people go into cardiac arrest, they quickly become acidemic and accumulate CO, in the blood. As CPR and ventilation begin, the CO₂ is offloaded in the alveoli, expired, and subsequently sensed by the end-tidal capnometer. End-tidal CO, has been shown to correlate with cardiac output even in low-flow states such as CPR, making it an indicator of CPR quality.9 With everv increase of 10 mm in the depth of chest compressions, there is a 1.4 mmHg increase in the end-tidal value, allowing you to monitor whether the compressor is becoming fatigued and in need of relief.¹⁰ That being said, ongoing CPR should produce at least a minimal end-tidal capnogram waveform; if you see a flat capnogram, check your advanced airway for correct positioning.

The survival rates of out-of-hospital cardiac arrest are dismal, with estimations of less than 6 percent.¹¹ End-tidal can help provide some prognostication during an active resuscitation. Having an end-tidal value less than 14.3 mmHg after undergoing 20 minutes of ACLS was predictive of not achieving return of spontaneous circulation (ROSC), with a sensitivity, specificity, positive predictive value, and negative predictive value of 100 percent.¹² In a separate study where the end-tidal value at three minutes post endotracheal intubation was measured, a value 10 mmHg was also associated with a low chance of ROSC.13 In a review of 23 observational studies related to the relationship between EtCO, and cardiac outcomes, it was emphasized that EtCO, cannot be used as a single prognostic factor and other characteristics of the arrest must be con-

sidered; however, the correlation between low EtCO, and low probability of achieving ROSC was again recognized. Larger organizations, such as the International Liaison Committee on Resuscitation and the American Heart Association (AHA), have agreed that low EtCO, suggests a low probability of ROSC, with the AHA specifically mentioning values below 10 mmHg.^{7,14} In a more positive light, an abrupt increase of >10 mmHg in EtCO, was associated with ROSC and shown to be a highly specific (97 percent) but not sensitive (33 percent) marker.¹⁵ During CPR, when you are typically left with very little information, end-tidal capnometry provides you with quick, reliable information that can actively guide your next resuscitation.



18 ACEPNOW DECEMBER 2016



Conclusion

End-tidal capnometry wasn't meant to be kept in the stable and only used for the occasional procedural sedation. Try taking it out for a ride on your next shift, whether it's for screening the next hyperglycemic patient for diabetic ketoacidosis, monitoring for disposition of a wheezing asthmatic, resuscitating or terminating an active cardiac arrest, or confirming placement of an NGT. Remember to use caution in patients with underlying lung disease such as chronic obstructive pulmonary disease. Throughout each shift, emergency physicians make critical decisions by pooling myriad data points from the history, physical exam, labs, imaging studies, and many other variables that we may subconsciously consider. End-tidal capnometry provides us with another data point that can aid in these crucial choices and requires few resources, money, or time.

UTILIZING ETCO₂ IN THE ED

- 1. EtCO, ≥36 mmHg to rule out DKA in hyperglycemic patients
- 2. Place NG/OGT to 30 cm, connect EtCO, if normal waveform then stop advancement;
- if no waveform, then continue advancement
- 3. Terminate CPR if EtCO, <10 mmHg during active CPR
- 4. Change hand position or compressor if worsening EtCO₂ or waveform during active CPR

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CODING WIZARD



NAVIGATE THE CPT MAZE, OPTIMIZING YOUR REIMBURSEMENT

Editor's Note: Cutting through the red tape to make certain that you get paid for every dollar you earn has become more difficult than ever, particularly in our current climate of health care reform and ICD-10 transition. The ACEP Coding and Nomenclature Committee has partnered with ACEP Now to provide you with practical, impactful tips to help you navigate through this coding and reimbursement maze.

Document Thoroughly to Receive Correct Level of Service Payment

by MIKE LEMANSKI, MD, FACEP, FAAFP

Question: I know there are five levels of service for emergency department E/M codes, but what really determines the level?

Answer: In addition to the nature of the complaint, the complexity of your medical decision making (MDM) is paramount. How extensive was your differential diagnosis? After you performed the history and physical examination, did you need to do any additional work-up? The results of

lab tests and your review or interpretation of ECGs and X-rays should all be documented. Be sure to document any bedside ultrasounds and any discussions you have with the radiologist about the results of CT scans. All of these add to the complexity of the data reviewed. Did you need to consult a specialist? This would also add to the complexity of the care delivered. If the patient was treated with IV fluids or received any medications, documenting the treatment and the patient's response helps show the risk of complications. Of course, your documentation of the history and physical examination needs to be complete and accurate. More information about getting credit for additional work-up can be found at the ACEP FAQ page under the Medical Decision Making and the Marshfield Clinic Scoring Tool FAQ at www.acep.org/Physician-Resources/Practice-Resources/Administration/Financial-Issues-/-Reimbursement/Medical-Decision-Making-And-The-Marshfield-Clinic-Scoring-Tool-FAQ/. O

Brought to you by the ACEP Coding and Nomenclature Committee.

DR. LEMANSKI is associate professor of emergency medicine at Baystate Medical Center/Tufts University School of Medicine in Springfield, Massachusetts, and chair of the ACEP Coding and Nomenclature Committee.

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by JAMES J. AUGUSTINE, MD, FACEP

e are excited to let you know that the board approved a major renovation of the emergency department," the CEO announces at your department meeting. "So your job is to design it correctly."

This is a once-in-a-lifetime experience for most emergency physicians. What elements will result in the "best design"?

The Emergency Department Benchmarking Alliance (EDBA), a not-for-profit organization that offers resources to ED leaders, has developed a data resource that will be beneficial for the project. In addition, ACEP recently published a book related to this topic, *Emergency Department Design: A Practical Guide to Planning for the Future, 2nd Edition*.

The design of the ED physical environment is critical to patient and staff safety as well as to satisfaction. Each department has a unique blend of patient needs, processes, and environments that design characteristics can help to optimize. It is critical for each emergency department to be designed as the front porch and front door to the hospital for the next 30 years, which is a typical lifespan for an emergency department.

The ability to respond to critical patient needs is the primary driver of ED design. The design should provide a safe workspace for staff and be built around the information technology commonly used in most emergency departments. The trend in the design of emergency departments is to increase the physical footprint of the department and create more patient care spaces. In our report two years ago, the EDBA found that across most ED volume cohorts, the average is about 1,550 visits per patient care space.¹ The pediatric-serving emergency departments, which flow patients more quickly, averaged about 1,900 visits per patient care space.

The EDBA data survey now has 10 years of data to report, and the trends are apparent. Emergency departments are being designed with more square footage and more patient care areas. These design principles allow the staff to manage a patient population that is older, is more medically complex, is of higher acuity, and experiences longer lengths of stay. The 10-year trend shows a decrease in the average utilization of patient care space, from about 1,723 visits to about 1,500 visits (see Table 1, p. 27). Emergency departments serving a higher mix of children and lower overall patient acuity can be served with fewer patient care spaces. The relative square footage has also increased, with average space utilization decreasing in 10 years from 3.9 to about 3 patient visits per square foot.

We have noted that more boarding increases the need for both ED space and ED care spaces. An increasing number of emergency departments are building patient care areas for vertical patients. Emergency depart-**CONTINUED** *on page 27*

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The Emergency Group, Inc. (TEG) is a growing, independent, democratic group that has been providing emergency services at The Queen's Medical Center (QMC) in Honolulu, Hawaii since 1973. QMC is the largest and only trauma hospital in the state and cares for more than 65,000 ED patients per year. QMC opened an additional medical center in the community of West Oahu in 2014, which currently sees 50,000 ED patients annually.

Due to the vastly growing community in the West Oahu area, TEG is actively recruiting for EM Physicians BC/BE, EM Physicians with Pediatric Fellowship who are BE/BC and an Ultrasound Director. Physicians will be credentialed at both facilities and will work the majority of the shifts at the West Oahu facility in Ewa Beach, Hawaii.

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BENCHMARKING ALLIANCE | CONTINUED FROM PAGE 21

ments are providing care in chairs, recliners, and other upright spaces. It is not yet clear that patients benefit from vertical care.

For emergency departments using team triage models, spaces that can serve both for greeting and rapid care (some using recliners due to their flexibility) are a very effective use of square footage. Flow and patient throughput are enhanced by universal patient room design, and patient care rooms must be designed for infection control, noise reduction, and staff efficiency. At the department level, the design must facilitate ambulance reception, toileting availability, and cleanliness, and it must include space designed for the safe processing of mental health patients.

Frank Zilm, director of the Institute for **Health+Wellness** Design at the University of Kansas in Lawrence, has suggested that, beyond the appropriate size and number of patient care spaces, the next-generation emergency department has to address the risk to staff and patients from pandemics.

There is a tremendous emphasis on disaster preparedness. Frank Zilm, director of the Institute for Health+Wellness Design at the University of Kansas in Lawrence, has suggested that, beyond the appropriate size and number of patient care spaces, the nextgeneration emergency department has to address the risk to staff and patients from pandemics. This involves designing for early identification of at-risk patients, the ability to segregate flow through the emergency department, and the ability to create cohort quarantine zones within or adjacent to the emergency department. Another key element in emergency preparedness is the inclusion of a multipurpose utility dirty room (the MUD room) for patient decontamination and early care.

It is wise to plan for a clinical area that is sized around three visits per square foot and about 1,300 to 1,500 visits per care space. The projection of future demand and service requirements for emergency care is a daunting challenge. Designing for flexibility to accommodate new care models is critical. O

Reference

1. Augustine JJ. Emergency Department Benchmarking Alliance reports on data survey for next-generation ED design. ACEP NOW. 2014;33(8):22.

Table 1. EDBA Data Survey Volume Per	YEAR	VISITS PER PATIENT CARE SPACE, ALL EDs	VISITS PER PATIENT CARE SPACE, PEDIATRIC EDs	VISITS PER SQUARE FOOT, ALL EDs
Space and by Square Footage	2015	1,515	1,894	3.0
	2014	1,519	1,908	3.1
	2013	1,553	1,886	3.1
	2012	1,597	1,892	3.2
	2011	1,610	1,815	3.2
	2010	1,615	1,834	3.7
	2009	1,677	1,697	3.5
	2008	1,700	Not available	3.4
	2007	1,710	Not available	3.9
	2006	1,723	Not available	3.8

RAPIVAB™ (peramivir injection), for intravenous use Initial U.S. Approval: 2014

BRIEF SUMMARY OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use RAPIVAB safely and effectively. See full prescribing information for RAPIVAB.

-----INDICATIONS AND USAGE---

RAPIVAB is an influenza virus neuraminidase inhibitor indicated for the treatment of acute uncomplicated influenza in patients 18 years and older who have been symptomatic for no more than two days

Limitations of Use:

- · Efficacy based on clinical trials in which the predominant influenza virus type was influenza A; a limited number of subjects infected with influenza B virus were enrolled.
- · Consider available information on influenza drug susceptibility patterns and treatment effects when deciding whether to use.
- · Efficacy could not be established in patients with serious influenza requiring hospitalization

---DOSAGE AND ADMINISTRATION--

- Administer as a single dose within 2 days of onset of influenza symptoms. Recommended dose is 600 mg, administered by intravenous infusion for a minimum of 15 minutes.
- Renal Impairment: Recommended dose for patients with creatinine clearance 30-49 mL/min is 200 mg and the recommended dose for patients with creatinine clearance 10-29 mL/min is 100 mg.
- Hemodialvsis: Administer after dialvsis.
- RAPIVAB must be diluted prior to administration. See the Full Prescribing Information for drug compatibility information.

--DOSAGE FORMS AND STRENGTHS Injection: 200 mg in 20 mL (10 mg/mL) in a single-use vial.

--CONTRAINDICATIONS-

Patients with known serious hypersensitivity or anaphylaxis to peramivir or any component of RAPIVAB

---WARNINGS AND PRECAUTIONS--

- · Cases of anaphylaxis and serious skin/hypersensitivity reactions such as Stevens-Johnson syndrome and erythema multiforme have occurred with RAPIVAB. Discontinue RAPIVAB and initiate appropriate treatment if anaphylaxis or serious skin reaction occurs or is suspected.
- · Neuropsychiatric events: Patients with influenza may be at an increased risk of hallucinations, delirium and abnormal behavior early in their illness. Monitor for signs of abnormal behavior

-- ADVERSE REACTIONS-Most common adverse reaction (incidence >2%) is diarrhea

To report SUSPECTED ADVERSE REACTIONS, call 1-844-273-2327 or contact FDA at 1-800-FDA-1088 or www.fda.gov/medwatch

-----DRUG INTERACTIONS----

Live attenuated influenza vaccine (LAIV), intranasal: Avoid use of LAIV within 2 weeks before or 48 hours after administration of RAPIVAB, unless medically indicated.

- --- USE IN SPECIFIC POPULATIONS--
- Pregnancy: Use if benefit outweighs risk.
- · Nursing mothers: Caution should be exercised when administered to a nursing woman

Revised: 8/2016

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Important Safety Information

Rapivab[®] (peramivir injection) is indicated for the treatment of acute uncomplicated influenza in patients 18 years and older who have been symptomatic for no more than 2 days.

- Efficacy of Rapivab was based on clinical trials in which the predominant influenza virus type was influenza A; a limited number of subjects infected with influenza B virus were enrolled.
- Influenza viruses change over time. Emergence of resistance substitutions could decrease drug effectiveness. Other factors (for example, changes in viral virulence) might also diminish clinical benefit of antiviral drugs. Prescribers should consider available information on influenza drug susceptibility patterns and treatment effects when deciding whether to use Rapivab.
- Efficacy could not be established in patients with serious influenza requiring hospitalization.

Contraindications

Rapivab is contraindicated in patients with known serious hypersensitivity or anaphylaxis to peramivir or any component of the product. Severe allergic reactions have included anaphylaxis, erythema multiforme, and Stevens-Johnson syndrome.

Warnings and Precautions

 Rare cases of serious skin reactions, including erythema multiforme, have been reported with Rapivab in clinical studies and in postmarketing experience. Cases of anaphylaxis and Stevens-Johnson syndrome have been reported in postmarketing experience with Rapivab. Discontinue Rapivab and institute appropriate treatment if anaphylaxis or a serious skin reaction occurs or is suspected. The use of Rapivab is contraindicated in patients with known serious hypersensitivity or anaphylaxis to Rapivab.

- Patients with influenza may be at an increased risk of hallucinations, delirium, and abnormal behavior early in their illness. There have been postmarketing reports (from Japan) of delirium and abnormal behavior leading to injury in patients with influenza who were receiving neuraminidase inhibitors, including Rapivab. Because these events were reported voluntarily during clinical practice, estimates of frequency cannot be made, but they appear to be uncommon. These events were reported primarily among pediatric patients. The contribution of Rapivab to these events has not been established. Patients with influenza should be closely monitored for signs of abnormal behavior.
- Serious bacterial infections may begin with influenza-like symptoms or may coexist with or occur as complications during the course of influenza. Rapivab has not been shown to prevent such complications.

Adverse Reactions

The most common adverse reaction was diarrhea (8% Rapivab vs 7% placebo). Lab abnormalities (incidence \ge 2%) occurring more commonly with Rapivab than placebo were elevated ALT 2.5 times the upper limit of normal (3% vs 2%), elevated serum glucose greater than 160 mg/dL (5% vs 3%), elevated CPK at least 6 times the upper limit of normal (4% vs 2%) and neutrophils less than 1.0 x 10⁹/L (8% vs 6%).

Concurrent use with Live Attenuated Influenza Vaccine

Antiviral drugs may inhibit viral replication of a live attenuated influenza vaccine (LAIV). The concurrent use of Rapivab with LAIV intranasal has not been evaluated. Because of the potential for interference between these two products, avoid use of Rapivab within 2 weeks after or 48 hours before administration of LAIV unless medically indicated.

You are encouraged to report negative side effects of prescription drugs to the FDA. Visit www.fda.gov/medwatch or call 1-800-FDA-1088.

References: 1. Rapivab [package insert]. Durham, NC: BioCryst Pharmaceuticals, Inc; 2014. 2. Kohno S, Kida H, Mizuguchi M, Shimada J; S-021812 Clinical Study Group. Efficacy and safety of intravenous peramivir for treatment of seasonal influenza virus infection. Antimicrob Agents Chemother. 2010;54(11):4568-4574. doi:10.1128/AAC.00474-10.

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