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THE JOURNAL OF URGENT CARE MEDICINE®

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Toxicological Emergencies for the Urgent Care Physician

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LETTER FROM THE EDITOR-IN-CHIEF

Physician Mentoring: Making an Impact

"We make a living by what we get, we make a life by what we give."

- Winston Churchill



hysician mentoring sounds like an easy enough proposition. Who wouldn't jump at the chance to opine and proselytize, in a position of power, to a new employee who is looking to impress his/her boss? Indeed, you can say most anything you want, k of rebuttal or confrontation

with a very low risk of rebuttal or confrontation.

Even well-intentioned mentors tend to preach, encouraging their subjects with snippets, wisdom, and praise. "Flyins" I call them: Fly in for a quick speech, receive a befuddled nod of confirmation, then fly out. We consider ourselves to be experts, and therefore, all we need to do is share our expertise to qualify for mentoring.

Well, I hate to break the news to you, but expertise is not in and of itself mentoring. Mentoring is a more collaborative approach to impact positive change in others in support of professional, personal, and organizational development. It *depends on* expertise and experience, but only as a springboard for deeper work and reflection. It implies growth and maturation, not just solutions.

Mentoring can have a lifelong impact for both the "mentoree" and the "mentorer," and can lead to a greater level of satisfaction and purpose for both.

So, what are the key characteristics of exceptional mentors? They are...

Available

Availability breeds confidence and trust, which encourages an openness that allows for an honest exploration of strengths and weakness, as well as for opportunities to provide adequate praise to buffer relevant concerns.

Reflective

- The ability to share your personal experiences, including the missteps, creates a disarming environment.
 - This allows the mentor to use insight and experience to help problem-solve effectively, and helps a new

employee gain perspective

Influential

A good mentor translates a position of authority into persuasive influence, relying on the ability to change behavior without bullying and to be confident, but not cocky.

Accepting

- Welcomes new ideas and personalities and identifies opportunities within the practice to take advantage of same.
- Willing to challenge their own ideology in an effort to learn from others.
- Not trying to create a homogenous practice.

Collaborative

- Works with a new employee to identify strengths and weaknesses and encourages their participation in how to address both.
- Offers opportunities for a new employee to get involved at a leadership level so that employee feels empowered.

Observant

Identifies potential problems before they become crises. Intervenes proactively.

New physicians are reluctant to publicly declare their desire for mentoring; it sounds weak. Nonetheless, a practice looking to engage new employees—and retain them for the long term—should assume the need exists, and provide accordingly. You just might be surprised to find that the return is more profound than the investment.

emit

Lee A. Resnick, MD Editor-in-Chief JUCM, The Journal of Urgent Care Medicine

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CLINICAL

11 Toxicological Emergencies for the Urgent Care Physician

Poisonings are second only to motor vehicle accidents among causes of accidental death in the U.S. Certain toxicological emergencies are most likely to present to urgent care. Are you and your staff prepared to manage them? By Michael L. Epter, DO, FAAEM and Alicia Pilarski, DO

BOUNCEBACKS

23 The Case of a 51-year-old Man with Back Pain

A presenting complaint of new-onset back pain might raise a host of red flags in certain patients. But what about an otherwise healthy man with no significant medical history? How does one identify the concerning

patient without over-testing every person who walks in the door? By Jill C. Miller, MD, and Michael B. Weinstock, ME

IN THE NEXT ISSUE OF JUCM

Pain or swelling in the scrotum area can indicate a surgical emergency. Due to the location of the problem, it's not unusual for the patient's first stop to be an urgent care center. Fast and thorough assessment is especially important in younger men and boys.

WEB EXCLUSIVE

In the Beginning: Doc's In ERgent Care in Clermont, FL

How does a successful businesswoman with particular expertise in marketing ultimately find herself, several years post-medical school, opening an urgent care center? You can read Dr. Cheryl Durstein Decker's story only at *www.jucm.com*.

By Sally Michael

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FROM THE EXECUTIVE DIRECTOR

Astonishing

LOU ELLEN HORWITZ, MA

eeeeeeere's fall! School physicals, flu season, indoor home improvement project injuries.... Can slipping on ice and minor frostbite be far away? Ah, the busy season!

From what I am hearing from all of you across the country, 2010 has been a mixed bag, thus far. Some centers are so busy they can hardly breathe, while others had a quiet first quarter since influenza was comparatively tame, and still weren't sure what the summer would bring as of our spring convention.

Some centers are opening their fourth (or 24th) locations, and the principals haven't seen their families in daylight hours for months. Others are facing the challenge of being the first urgent care center in their area, and are struggling to educate the community on what urgent care is all about.

It's hard to know who has it better.

Most of you are also fighting misconceptions about what you do, who you are "stealing" patients from, whether your center has any impact on the ED or not, the shifting sands of state regulations, and mixed messages (to both you and your patients) from insurance companies.

Everyone wants to know what "healthcare reform" is going to mean, and no one does. And yet, urgent care is in the news now, every single day, so...we must be doing some things correctly.

But how did we get here?

Before you think you are in for a "history of urgent care" lecture (which cannot be done anyway, as our origins are shrouded in mystery!) let me be clearer: Why are you and your staff (and your families, by extension), and all of us in the UCAOA offices, swimming in this urgent care pool at all?

Did you know that the Statue of Liberty was almost not completed because the U.S. couldn't raise our half of the funds to pay for the base and pedestal? An astonishing



Lou Ellen Horwitz is executive director of the Urgent Care Association of America. She may be contacted at *lhorwitz@ucaoa.org*.

"In the day-to-day, you have to find it in the one-to-one."

120,000 contributions, most for under \$1 (yes, that's one dollar) finally combined to reach the necessary \$100,000.

Before you dismiss that little anecdote as a complete non sequitur, let me explain; the story strikes me as similar to how urgent care has gotten where it is today. There are a lot of you, and most of your businesses are small. *But*, you are willing to take what you have and put it toward the common good, and little by little, fairly quietly, you are building something that is starting to get noticed by the rest of the country. That's a pretty cool thing to be a part of.

It can be hard to see from your individual positions, though. Especially at the end of a long day, or after a tough phone call, or a frustrating negotiation with a system seemingly designed to thwart your success.

At one end, you are that brand new center and just hoping against hope that someone is going to pull into your parking lot. At the other end you are just hoping the phone will stop ringing long enough for you to catch up on your email or charting. Yet, you continue.

Somewhere in each of you there is a desire to be part of something unique, important, and impactful. In the day-today, you have to find that in the one-on-one. Making a difference in the life of a patient—be it in the exam room, or with a billing question, or via a constructive chat with one of your staff. All those little differences are your contributions to our Statue of Liberty.

Don't stop. Collectively, you are starting to get noticed for what you have quietly been building for years. And one day, it is going to be astonishing (if it isn't already).

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A n 8-year-old boy. A 55-year-old man. A 24-year-old woman living at home with her parents. A 22-year-old college student. Disparate patients with one thing in common: Each is suffering the effects of intoxication after ingesting substances readily at their disposal. Those substances may be medications commonly found in the average home, or they may be recreational drugs.

Once these patients walk through your door, you may not have much time to figure it out, and they may not be able to assist you in gathering evidence. Quick action on your part, whether that means acute decontamination or a quick workup while waiting for transport to the ED, can be the determining factor in the patient's survival.

In Toxicological Emergencies for the Urgent Care Physician (page 11), **Michael L. Epter, DO, FAAEM** and **Alicia Pilarski, DO** walk us through



the scenarios described above, as well as a couple more.

Dr. Epter is an assistant professor and the director of the Emergency Medicine Residency Program at the University of Nevada, as well the president of the Nevada Chapter of the American Academy of Emergency Medicine. In addition to resident and medical student education, he has a strong interest in orthopedics and toxicology.

Dr. Pilarski is an assistant professor in the Department of Emergency Medicine at the Medical College of Wisconsin, Froedtert Hospital, Milwaukee, WI, as well as the chair of the Membership Committee for the Young Physicians Section of the American Academy of Emergency Medicine. She shares Dr. Epter's commitment to resident and medical student education, and is also keenly interested in critical care in the ED and management of syncope.



A case-based approach is also employed in the latest installment of the Bouncebacks series. This month, **Jill C. Miller, MD** and **Michael B. Wein**-

stock, MD recount the case of a 51-year-old man who presented with back pain. There's nothing out of the ordinary in his history, so where would you draw the line between a thorough exam and unnecessary tests? Dr. Miller is senior clinical instructor at Case Western Reserve University School of Medicine and is board certified in internal medicine. She practices urgent care with University Hospitals Medical Practices in Cleveland, OH. Dr. Weinstock is clinical assistant professor of emergency medicine at The Ohio State University School of Medicine, as well as a practitioner in the Mt. Carmel St. Ann's Emergency Department in Columbus, OH.

Finally, in an article available only at *www.jucm.com*, **Sally Michael** shares the story of a physician whose experience in business before answering the call to practice medicine proved to be invaluable asset in founding Doc's In ERgent Care in Clermont, FL.

The article is part of the In the Beginning series, in which we look at the lessons learned by entrepreneurs who have taken the initiative to start up a new urgent care center.

Ms. Michael is communications director for The Lohman Group, Inc. in Falls Church, VA. A lifelong communications professional, she also directed the largest HIV/AIDS program in Virginia in the late 1990s.

Also in this issue:

Nahum Kovalski, BSc, MDCM reviews new abstracts on diagnostic imaging for head injury, causes and treatment of cellulitis, the use of alteplase after onset of ischemic stroke, the American Academy of Pediatrics' revised clinical report on head lice, and other current topics relevant to urgent care.

John Shufeldt, MD, JD, MBA, FACEP manages to find the rare space where the famed Miracle on the Hudson flight intersects with a Van Halen concert, producing an important lesson on the value of checklists and preparation in the urgent care center.

Frank Leone, MBA, MPH offers rationale for his belief that "just say no" is a good policy where cold calls to pitch urgent care occupational medicine services are concerned.

David Stern, MD, CPC illuminates the new changes in the ICD-9 code set.

As we begin our fifth year of publication, we're as eager as ever to expand our roster of outstanding contributors. If you'd like to be one of them, drop a quick note to our editor-in-chief, **Lee A. Resnick, MD**, at *editor@jucm.com*.

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A Note of Thanks to Our Peer Reviewers

verybody knows the quarterback's name; he throws the touchdowns and gets the endorsements. Take away the linemen who make sure he has the time to deliver the ball before being crushed by a 350-pound defensive tackle, though, and you'd be hard pressed to find willing candidates to fill the position.

JUCM readers know the names of all the authors who contribute articles—they're literally right there in black and white but there's a growing list of clinicians who contribute behind the scenes by participating on our peer-review panel.

The peer-review process is not a complicated one, but it's an integral part of our mission to publish content that is relevant to the way you practice, clear in the way procedures are described, and free of even a hint of commercial bias. Participation requires

clinical acumen, critical thinking, and a desire to help boost the level of discourse in the urgent care arena without being credited with an assist.

We do not share with our authors or readers the names of individuals who have reviewed a particular article (nor do we share the names of authors with the reviewers until publication; the process works best if it's "double-blind"). However, we would be remiss if we did not publicly thank our reviewers—all of whom are busy practitioners and/or academics who have no trouble filling the hours in a day—for making time to contribute in this way.

We are grateful to the following individuals for sharing their time and expertise—some on more than one occasion—in reviewing articles that appeared in Volume 4 (October 2009 through September 2010) of *JUCM*:

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Toxicological Emergencies for the Urgent Care Physician

Urgent message: Several specific toxicological emergencies are most likely to be encountered in the urgent care setting. Prompt recognition of their clinical presentation, understanding the pathophysiology/ natural disease progression, and initiation of treatment are critical factors in decreasing morbidity (and potential mortality) in these cases.

Michael L. Epter, DO, FAAEM and Alicia Pilarski, DO

Introduction

eginning in 2004, poisonings rank second to motor vehicle accidents as the leading cause of accidental death in the U.S., with unintentional ingestions constituting the largest component of poisoning deaths.¹ The most common fatal ingestants included sedative hypnotics/antipsychotics, opioids, antidepressants, cardiovascular drugs, acetaminophen (with or without combinations), alcohols, and street drugs/stimulants.²

Utilizing a case-based format, this article will seek to:

- formulate general man- © istockphoto.com agement guidelines for evaluation and treatment of toxicologic emergencies
- appraise evidence-based recommendations for acute decontamination

(e.g., asymptomatic and benign to fatal), management of these patients in the urgent care setting needs to prompt and selective, with the evaluation of all patients immediately upon arrival.

- describe and differentiate among common ingestions and drugs of abuse
- demonstrate the role of initial laboratory tests and radiographs in the management of these patients
- provide clinical pearls and mnemonics to aid in identification of commonly encountered poisons.

Urgent Care Management

Because the natural history of ingestions has a wide and variable clinical presentation with dynamic changes in a patient's status



Table 1. Acutely	Poisoned Patient: General Management Guidelines				
A-B-C-D-E-F	 A: Airway – ensure airway is protected B: Breathing – provide supplemental oxygen as needed and ensure adequate ventilation C: Circulation – ensure adequate perfusion; initiate IV therapy with NS bolus (20 cc/kg in children); multiple boluses may be required for hypotensive patients D: Decontamination – administer activated charcoal (AC) for most ingestions unless otherwise contraindicated (1g/kg; 60-90g for most adults) E: ECG – evaluate for any dysrhythmias; treat as appropriate F: Fingerstick – assess for hypoglycemia; treat if indicated 				
Monitor	Place patient on cardiac monitor; assess vitals frequently				
Seizures	Avoid secondary insult (e.g., aspiration, trauma), provide supple- mental oxygen, benzodiazepines if available				
Miscellaneous	 Expose patient to assess for secondary injury (e.g., trauma) Assess for suicidal intent Administer specific antidote if available and the patient is clinically toxic (e.g., naloxone for opiate intoxication, sodium bicarbonate for patients intoxicated with tricyclic antidepressants) 				

emy of Pediatrics, which recommended against use of IS as a routine home treatment following ingestion, as well as for disposal of ipecac in the home.⁴

There are several complications involved with evoked emesis, including aspiration, airway compromise, and injury to the esophagus.

A 2005 position paper from the American Academy of Clinical Toxicology/European Association of Poisons Centres and Clinical Toxicologists stated that IS should not be administered routinely to poisoned patients and to consider ipecac only in an alert, conscious patient who has ingested a potentially toxic amount of a poison within the past 60 minutes.⁵

Absolute contraindications include nontoxic/acid/alkali/hydrocarbon/ sharp object ingestions, as well as use in patients who are altered/comatose,

This assumes management is appropriate for a given urgent care center, and will vary according to availability of resources (e.g., lab support, staff personnel).

Patients who present with or develop altered mental status (e.g., Glasgow Coma Scale score ≤14, confusion, and agitation), abnormal vital signs, suicidal ideation, repeated vomiting, abnormal ECG findings, or those who will require extended observation require immediate transfer to the emergency department.

In contrast, most alert, stable patients can remain in a clinic setting safely as management decisions are being made.

Consultation with a local/regional Poison Control Center (1-800-222-1222) can aid in the evaluation/disposition of these patients.

For those patients with confirmed/suspected intentional ingestions, clinicians should make disposition decisions in coordination with a psychiatric consultant.

Acute Decontamination

Ipecac syrup

Ipecac syrup (IS) has, historically, been a mainstay of gastrointestinal (GI) decontamination. However, current literature does not favor the use of induced emesis due to the low benefit-to-risk ratio coupled with significant contraindications and adverse effects of its administration.³ This notion was furthered by the American Acad-

actively vomiting, have no protective airway reflex, have known/suspected increased intracranial pressure, are pregnant, within hypertensive crisis, and/or expected to deteriorate.

Activated charcoal

Activated charcoal (AC) is the most common method of GI decontamination (recommended dosage: 1 g/kg) in poisoned patients.⁶ Toxins absorb to AC in the small intestine and then are excreted.

Exceptions include alcohols, lithium, acids/alkalis, pesticides, hydrocarbons, iron, arsenic, and other small, ionized and water-soluble compounds.

In situations where multiple co-ingestants may be present (including corrosives), AC should still be given if there is a risk of systemic toxicity.

AC is contraindicated in patients who have no bowel sounds, risk for GI perforation/hemorrhage, active vomiting, loss of protective airway reflexes, or when endoscopic visualization is anticipated.

If a patient is obtunded, the airway must first be secured; then AC can be administered through an orogastric or nasogastric tube.

Avoid using cathartics mixed with the activated charcoal, since electrolyte imbalances can occur.

General management guidelines for the acutely poisoned patient are described in Table 1.

Case #1

An 8-year-old male presents with a complaint of blood-tinged vomiting. Mom was tending to her newborn baby when he began to develop these symptoms.

Iron toxicity

Iron overdose is a common and potentially fatal ingestion in two patient populations: children and expectant mothers.

Iron toxicity is common in children due to an iron tablet's close resemblance to candy, and the lack of recognition by the caretaker of iron as a poison.⁷ Pregnant females also are at higher risk for iron toxicity, given the recommendation of utilizing prenatal vitamins (which contain iron) to promote embryonic development.

The primary organ systems involved in iron toxicity include the gastrointestinal and cardiovascular

Table 2. Ferrous Salt–Iron Content Conversion Chart					
Salt	% elemental iron content				
Sulfate (most common preparation)	20%				
Gluconate	12%				
Fumarate	33%				
Children's chewable tablets	18 mg/tablet				

Table 3. Determination of Iron Toxicity Based on Elemental Iron Ingested				
Non-toxic	<20 mg/kg			
Mild to moderate toxicity	20-60 mg/kg			
Severe toxicity	>60 mg/kg			

system, but it can also affect other organ systems as a re-

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sult of metabolic acidosis.

Within the GI system, iron is directly toxic due to its corrosive effect on the stomach, which can lead to vomiting, diarrhea, and GI bleeding. Hepatotoxicity results from free radical production and oxidative damage.⁸

In the cardiovascular system, iron directly damages blood vessels, causes vasodilatation, and blocks oxidative phosphorylation.

In addition, iron is a negative inotrope, and directly toxic to the myocardium. Metabolic acidosis is produced through lactic acid production (secondary to interference of oxidative phosphorylation and hypotension) and through the conversion of iron from the ferrous state (Fe²⁺) to the ferric state (Fe³⁺), which releases a hydrogen ion (see **Table 2**).⁷

To calculate the amount of elemental iron ingested, the following formula can be utilized:

(# tabs ingested) x (mg iron
$$\frac{salts}{tab}$$
) x (% elemental iron)
patient weight (kg)

For example, consider the 8-year-old boy described in this case. He weighed 30 kg, and ingested 20 tablets of 325 mg ferrous sulfate tablets. Therefore, applying the formula would yield the following:

$$\frac{(20) \times (325) \times (20\%)}{30 (kq)} = 43 \frac{mg}{kg}$$
 of elemental iron ingested

(Note, however, that if the patient is pregnant, prepregnancy weight should be used in the calculation.⁷)

In general, level of iron toxicity can be determined by the amount of elemental iron ingested (**Table 3**).

Other diagnostic modalities that can assist in iron toxicity include an electrolyte panel to evaluate for anion gap metabolic acidosis (AGA) and abdominal radiographs to evaluate for radiopaque pill fragments. Radiographs can be helpful in the acute setting when the ingestion is unknown; however, their yield is inversely proportional to time from ingestion. A negative x-ray does not exclude a possible ingestion. (See **Table 4** for radiopaque compounds on x-ray).

Management follows standard protocol (see Table 1), and includes decontamination if other co-ingestants are suspected. Supportive management of hypotension with IV fluids and treatment of nausea and vomiting are the major cornerstones of initial management. Definitive management includes deferoxamine; a published consensus guideline recommends that all patients with \geq 4 episodes of vomiting, ingestion of 40 mg/kg of elemental iron, and/or suspected toxic ingestion should be immediately transferred to the nearest ED.⁷

The following questions should be answered if iron toxicity is suspected:

- 1. Any history of emesis?
- 2. How many episodes of emesis? (More than four episodes of emesis suggests systemic toxicity.)
- 3. What type of iron was ingested (sulfate, fumarate, gluconate) and in what form (e.g., tablet/liquid/ chewables)?

Case #2

A 55-year-old male presents with left shoulder pain after a lifting injury. He has been taking a pain reliever for the last two days without any relief. The patient presents to the clinic for a stronger pain medication, but also complains of "ringing in his ears" and nausea for the last few hours.

Salicylate toxicity

Salicylates (i.e., aspirin, methyl salicylate) are commonly used for analgesia, but also as antipyretic, anti-inflammatory, and anti-platelet agents, and are easily accessible to the general public.

Salicylate poisoning can be either acute or chronic in nature, and clinicians must maintain a high index of suspicion in these patients. In 2004, there were over 21,000 aspirin and non-aspirin salicylate exposures reported to U.S. poison centers. Of those, 43 cases resulted in death and 12,968 patients required hospital treatment.⁹

Systemic effects occur secondary to inhibition of oxidative phosphorylation, direct stimulation of the central respiratory center, GI irritation, and increased capillary and pulmonary endothelial permeability. This leads to the classic findings of a respiratory alkalosis with a metabolic acidosis, hyperventilation, GI effects (e.g., vomiting, GI bleed), hypotension, altered mental status, seizures, and kidney and liver damage.

Patients may also complain of tinnitus, which may be an early indicator of CNS toxicity.⁹ Chronic toxicity is most commonly seen in the elderly population due to declining renal function and utilization of multiple medications that may contain aspirin. It is characterized by a non-specific presentation, and can be confused with a sepsis syndrome, dementia/psychosis, and pulmonary edema.

Acute management is supportive (**Table 1**), and immediate transfer to an ED is indicated if >150mg/kg of aspirin is ingested, and/or the patient is clinically toxic.



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In addition to supportive treatment, the patient should be decontaminated with activated charcoal (1 g/kg). The patient will need immediate transfer to a higher level of care for the administration of sodium bicarbonate to help alkalinize the urine. This helps facilitate excretion of salicylate, while also helping to prevent absorption into organ tissues (i.e., the central nervous system [CNS]).

Diagnosis of salicylate toxicity must also include the possibility of co-ingestants. An electrolyte panel will show an AGA—specifically, a mixed respiratory alkalosis with metabolic acidosis; however, this is best displayed on ABG.

Glucose may be decreased and the patient may also have hyperkalemia and renal insufficiency/failure secondary to the direct and indirect effects on the renal system.

In chronic ingestions, a CBC may show anemia from an underlying GI bleed. A chest x-ray should be obtained if the patient has any respiratory symptoms and/or signs of pulmonary edema.

Obtain an ECG for any possible dysrhythmias, especially to evaluate for any changes related to hyperkalemia.

The following clinical questions should be answered if salicylate toxicity is suspected:

- 1. How much salicylate was ingested?
- 2. Does the patient have tinnitus?
- 3. Is the patient tachypneic (compensation for acidosis)?

Case #3

A 24-year-old female presents to the clinic after her mother found her lying on the ground and vomiting. The patient admits to taking a large quantity of pills that were found in the medicine cabinet.

Non-steroidal anti-inflammatory drug (NSAID) toxicity

Similar to aspirin, NSAIDs are widely available and utilized for a variety of conditions, rendering them common ingestants. Over 107,000 case mentions due to NSAID ingestions were reported to Poison Control Centers in 2008.²

Drug absorption is rapid and will produce effects within two hours of taking the medication.

The effects of NSAIDs are due to competitive inhibition of the cyclooxygenase enzyme involved in prostaglandin synthesis. In an overdose, these effects become exaggerated and eventually impair the GI, renal, hepatic, and central nervous systems.

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available at *www.jucm.com*; just click on the 2010/2011 Urgent Care Buyer's Guide button.Or, go directly to *www.jucm.com/buyersguide*.You'll find all the resources in this book (and then some), organized by category or searchable by key word. Patients most commonly present with GI symptoms (e.g., abdominal pain, vomiting, GI bleed) but can also have CNS depression and seizures, depending on the severity of ingestion and/or class of drug ingested (e.g., mefenamic acid).¹⁰

Patients with a history of GI bleed, peptic ulcer disease, and/or alcohol abuse are at greatest risk for developing an acute GI bleed—the most common cause of mortality in such cases. Elderly patients are also at higher risk for toxicity secondary to a decreased baseline renal function. An electrolyte panel may help to determine the baseline renal function, as well as any electrolyte abnormalities and AGA.

Most patients will improve with supportive care (**Table 1**), and ingestions of less than 100 mg/kg are unlikely to result in toxicity. The patient should be transferred if she is clinically toxic (e.g. GI, renal, hepatic, CNS dysfunction) or if reported ingestion of ibuprofen is >400 mg/kg, since the patient may require hemodialysis for definitive removal of the agent.¹⁰

The following clinical questions should be answered if NSAID toxicity is suspected:

- 1. How many pills were taken and what strength were the tablets?
- 2. Does the patient have any melena/bright red blood per rectum/hematemesis?
- 3. Does the patient have a history of baseline kidney dysfunction?

Case #4

A 30-year-old male presents complaining of nausea, vomiting, and abdominal pain. He states he took "some pills" last night with heavy amounts of alcohol in an attempt to commit suicide.

Acetaminophen toxicity

Acetaminophen (APAP) toxicity is one of the most common causes of potentially toxic ingestions, and alone or in combination therapy accounts for >161,000 case mentions reported to U.S. Poison Centers in 2008. Over half of all deaths attributed to analgesics are due to APAP.²

APAP toxicity is the leading cause of acute liver failure in western countries and contributes to the majority of admissions to liver transplant units.¹¹ Acetaminophen is metabolized primarily in the liver into sulfate and glucuronide conjugates, which are nontoxic and excreted in the urine.



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Table 4. Must-know Mnemonics					
Compound	Mnemonic				
Radiopaque substances on x-ray	CHIPES C: Calcium, cocaine, condoms, chloral hydrate H: Heavy metals I: Iron, iodide P: Psychotropics, bezoar E: Enteric coated pills S: Solvents (CCl ₄)				
Cholinergic toxicity	DUMBBELS D: Diarrhea U: Urination M: Miosis B: Bronchospasm, bronchorrhea B: Bradycardia E: Emesis L: Lacrimation S: Salivation, seizure				
Anion gap metabolic acidosis - AGA					
Anticholinergic toxicity	"Hot as a hare" "Blind as a bat" "Dry as a bone" "Mad as a hatter" "Red as a beet"	Hyperthermia Mydriasis Dry mucus membranes, decreased sweating Mental status changes Skin flushing			

Table 5. Predicted Effect Based on QRS Duration ¹⁵				
QRS Duration	Clinical effect			
<100 msec No significant clinical toxicity				
>100 msec	1/3 will have seizures; 14% ventricular dysrhythmias			
>160 msec	1/2 will have ventricular dysrhythmias			

A small percentage is metabolized by the cytochrome P-450 system into N-acetyl-p-benzoquinone imine (NAPQI), which is then reduced from this toxic form by glutathione into a nontoxic conjugate.

In overdose situations, the sulfation and glucuronidation pathways become saturated and the pathway shifts to the cytochrome P-450 system. This results in increasing amounts of NAPQI, which in turn depletes glutathione stores. Once the glutathione supply is depleted, the NAPQI compound becomes abundant and causes intracellular damage, primarily in hepatocellular cells.¹¹

There are four stages of acute APAP toxicity:

- 1. 0-24 hours: GI irritation (e.g., nausea, vomiting, abdominal pain)
- 2. 24-48 hours: resolution of GI symptoms with elevation of liver function tests
- 3. 48-96 hours: severe hepatic dysfunction (coagulopathy, acidosis, hypoglycemia, cerebral edema, and death can occur in this stage)
- 4. 5-14 days: recovery.

In patients with APAP ingestion, the most reliable time of ingestion must be determined. If unclear, the earliest time of ingestion should be utilized or corroborated with others (e.g., family). This will help with the assessment of the need for antidote administration when the patient is transferred to the ED.

Treatment of acute APAP toxicity involves supportive management (**Table** 1), as well as decontamination with charcoal and its antidote, N-acetylcysteine (NAC). NAC is a precursor for glutathione, which helps reduce NAPQI to a non-toxic substance and decrease hepatotoxicity.

In patients with a suspected or confirmed APAP ingestion (≥150 mg/kg), immediate transfer to the ED should be initiated, since lab testing and possible NAC administration may be warranted.

Because APAP is a common co-ingestant and has potentially devastating he-

patotoxic effects, APAP ingestion should be considered in all patients presenting to urgent care centers with drug ingestion of any kind. Rates of potentially hepatotoxic levels without history of ingestion have been found to be 0.3% even in patients without history of ingestion.¹²

Subacute/chronic toxicity should also be suspected in those who have risk factors for hepatotoxicity (e.g., EtOH consumption) and consume >4g/d since APAP is a component of numerous medications.¹³

The following clinical questions should be answered if acetaminophen toxicity is suspected:

- 1. What time did the ingestion occur?
- 2. How much acetaminophen was ingested (toxic dose in both children and adults is ≥150mg/kg)?
- 3. What form of acetaminophen was ingested (e.g., tablet/liquid/chewable/sustained release)?

Case #5

A 65-year-old female with a history of chronic neuropathy presents after seizure. Her husband reports she has had episodes of confusion and sometimes takes extra doses of her medications.

Tricyclic antidepressants

Antidepressants are the third most common cause of fatalities due to ingestion; tricyclic antidepressants (TCAs) account for >11,000 case mentions to U.S. Poison Centers.² The resurgence of TCAs may in part be due to treatment of neuropathies, chronic pain, and refractory depression.

The mechanisms of action for TCAs are extensive; in an overdose, these effects are enhanced and lead to potentially deadly consequences, including catecholamine depletion and relative hypotension, anticholinergic effects, seizures, coma, and cardiac dysrhythmias/conduction disturbances.¹⁴

Management of these patients includes supportive care (**Table 1**), with emphasis on fluid resuscitation for hypotension. An ECG should be obtained immediately to evaluate for widening of the QRS, since this can predict the effects of the TCA poisoning better than serum concentrations.

Other findings on ECG include a terminal R wave in aVR >3mm, and R/S >0.7 in aVR,¹⁶ though life-threatening complications can occur in the absence of ECG abnormalities.

AC should be administered, and the patient should be started on sodium bicarbonate (1 to 2 mEq/kg) if the QRS measures >100 msec on ECG (**Table 5**).

These patients can decompensate rapidly; any suspicion of TCA ingestion in patients who are symptomatic should prompt immediate transfer to an ED. Benzodiazepines should be administered in patients who have seizure activity.¹⁴

The following clinical questions should be answered if tricyclic antidepressant toxicity is suspected: 1. How many tablets were ingested?

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- 2. Does the patient have any EKG changes?
- 3. Is there any seizure activity?

Case #6

A 22-year-old male was at a party in college and was found by some friends to be having hallucinations and acting inappropriately. The patient appears profusely diaphoretic and febrile. The patient had taken some "tablets" at the party.

Ecstasy (MDMA) ingestion

Ecstasy is an amphetamine-derived compound that is used primarily for its euphoric effects by teenagers and young adults. It works primarily by increasing the release of dopamine, norepinephrine, and serotonin while also inhibiting their re-uptake. This results in a sympathomimetic response which can produce the following effects:

- tachycardia
- hyperthermia
- anxiety
- diaphoresis
- cardiovascular instability.

In addition, serotonin syndrome can develop due to the increase in circulating serotonin. Symptoms of serotonin syndrome include CNS effects (e.g., altered mental status, hallucinations), autonomic effects (e.g., diaphoresis, hypertension, tachycardia), and neuromuscular instability (e.g., myoclonus, hyperreflexia), ultimately leading to life-threatening hyperthermia.¹⁷

Serotonin syndrome can be a life-threatening complication of ecstasy and drugs of abuse such as LSD or cocaine, as well as many commonly used over-the-counter drugs (e.g., Robitussin) and prescription medications (e.g., SSRIs, monoamine oxidase inhibitors, lithium). Having a high degree of suspicion is needed to help diagnose this clinical syndrome.

The patient should be transferred to the ED if any vital signs are abnormal (e.g., tachycardia, hypertension, and fever), or if you find muscle rigidity, seizures, and/or changes seen on ECG.

Primary treatment of ecstasy (stimulant) ingestion is supportive; however, management is focused on fluid resuscitation, prevention of seizures with benzodiazepines, and cooling the hyperthermic patient.

If the patient has overdosed, AC can be given to help increase absorption.

A secondary concern with ecstasy ingestion is rhabdomyolysis, which can lead to electrolyte abnormalities (e.g., hyperkalemia, hypocalcemia), cardiovascular compromise, renal insufficiency/failure, and death. Treatment of hyperthermia requires aggressive cooling measures to help decrease core temperature. Cooling modalities such as a cool mist fans, wet blankets, ice packs to the groin and axillae, and infusion of cool normal saline can help with the patient's temperature while awaiting transfer.

The following clinical questions should be answered if ecstasy (MDMA) ingestion toxicity is suspected:

- 1. Does the patient have a fever and/or sympathomimetic features?
- 2. Does the patient have myoclonus and/or hyper-reflexia?
- 3. What type, amount, and route of medication ingested?

Summary

The ability to quickly identify the substance and amount ingested by a patient who appears to be suffering the effects of toxicity is critical to quick action and subsequent positive outcomes. Typically, such patients can be treated effectively in the urgent care center, with transfer or follow-up referral employed as needed.

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Bouncebacks

The Case of a 51-year-old Man with Back Pain

In Bouncebacks, which appears periodically in JUCM, we provide the documentation of an actual patient encounter, discuss patient safety and risk management principles, and then reveal the patient's "bounceback" diagnosis.

Cases are adapted from the book Bouncebacks! Emergency Department Cases: ED Returns (2006, Anadem Publishing, www.anadem.com; also available at www.amazon.com and www.acep.org) by Michael B. Weinstock and Ryan Longstreth. The book includes 30 case presentations with risk management commentary by Gregory L. Henry, past president of The American College of Emergency Physicians, and discussions by other nationally recognized experts.

Jill C. Miller, MD and Michael B. Weinstock, MD

A 51-year-old Man with Back Pain

ost new third-year medical students can recite the "red flags" of back pain: extremes of age, fever, history of cancer, history of trauma, failure to improve after one month of therapy.

Few would fail to consider metastatic disease in a 64-yearold woman with a history of breast cancer and new-onset low back pain, but what about the 51-year-old male without a significant past medical history?

The following case forces us to consider some important questions: Can we effectively triage urgent care patients without an onslaught of unnecessary tests? Can we tease out the concerning patients and avoid missing life-threatening diagnoses?

Initial Visit

(Note: The following, as well as subsequent visit sum-

maries, is the actual documentation of the providers, including punctuation and spelling errors.)

CHIEF COMPLAINT: Back pain

HISTORY OF PRESENT ILLNESS (at 08:50):

This is an otherwise healthy 51 y/o male who presents with a three to four week history of waxing and waning lower back pain. He denies any definite injury prior to symptom onset. He denies saddle paresthesias, bowel or bladder incontinence, weakness or numbness in the arms or legs. No fever or IV drug abuse. No prior back surgery. No meds prior to arrival. No fever, vomiting, chest pain, dysuria, urinary

frequency, paresthesias.

PAST MEDICAL HISTORY/TRIAGE:

No private physician

Vital Signs								
Time	Temp (°F)	Rt.	Pulse	Resp	Syst	Diast	Pos	Pain Scale
08:15	96.8	Т	88	18	164	107	8	8

No known allergies.

Meds: The patient is not taking medications at this time No significant medical history.

No significant surgical history.

EXAM (at 08:52)

General: Well-appearing; well-nourished; A&O X 3, in no apparent distress

Head: Normocephalic; atraumatic.

Nose: The nose is normal in appearance without rhinorrhea

Abd: Non-distended, non-tender, soft, without rigidity, rebound or guarding

Back: There is pain with palpation musculature low back. No midline cervical/thoracic/lumbar sacral tenderness to palpation = + lipoma on lower T-spine

Neuro: Strength 5/5 for flexion and extension bilateral lower extremities, patellar DTR's normal X2, straight leg raise negative X2, sensation grossly intact bilateral lower extremities. No evidence of urinary incontinence

PROGRESS NOTES (at 09:05):

His blood pressure remained 160/100 on recheck. He has no chest pain, shortness of breath, or lateralizing weakness or paresthesias. I suspect this blood pressure elevation is due to acute pain. I have given him instructions on blood pressure and he is to follow-up with his physician in the next few days for a recheck of his blood pressure. He received ibuprofen 600mg at 09:04.

PROCEDURES:

Urine dip stick: WNL except: Trace protein

DIAGNOSIS:

LBP (Low back pain)

DISPOSITION:

Aftercare Instructions for LS strain and elevated blood pressure, prescriptions for ibuprofen and vicodin. Patient left the ED at 09:14.

Discussion of Documentation and Risk Management Issues at Initial Visit

Error 1: Inadequate history.

Discussion: This history is an argument, a way to build your case to support the diagnosis. Though this may seem backward (the history *really* is about collecting data and then forming a diagnosis based on the evidence), if you are going to diagnose all back pain patients as having a strain, at least try to have the history

to support your diagnosis.

This history lacks most of the basic supporting evidence. There is no mention of exacerbating or relieving factors; in fact, this true history only describes two elements: duration and lack of mechanism. This history is much more of a review of symptoms than a history at all. **Teaching point:** When diagnosing a back strain, document if the pain is worse with motion.

Error 2: Lack of consideration of serious causes of back pain.

Discussion: If a back pain patient is presented at a morbidity and mortality conference, the audience would focus on the most serious possible causes:

- 1. *Epidural compression syndrome*. Is there an abscess or mass pressing on the cord which could result in paralysis? Is there a massive midline disk herniation? Surprisingly, the most sensitive historical factor is urinary retention, *not* urinary incontinence. Risk factors include history of intravenous drug use and/or fever (abscess) or weight loss (mass).
- 2. *Abdominal etiology.* Is there epigastric pain representing pancreatitis or an ulcer? Could this be pain referred from an ovarian cyst/abscess/torsion? How about retrocecal appendicitis?
- 3. *Is there an impending vascular emergency?* Though this patient is a bit young, the classic missed diagnosis in a back pain patient is abdominal aortic aneurysm (AAA) or ruptured AAA, often attributed to low back strain or stone (ureterolithiasis). The classic triad of back pain, hypotension, and pulsatile abdominal mass is present less than half the time, but this diagnosis needs to be considered in all back pain patients over the age of 50.

Teaching point: Think worse first.

Error 3: The patient was not informed of diagnostic uncertainty.

Discussion: I try to be as confident as possible, never letting a patient know I have any doubt. I give all my patients definitive diagnoses and prescribe meds for everyone. If I don't know their diagnosis, I just make one up. Patients love me! Recently, however, I was surprised when I told a patient I *thought* they had a certain diagnosis but that if their symptoms changed or persisted they would need to return. They did return, were correctly diagnosed, and loved me even more.

Teaching point: Inform patients when there is diagnostic uncertainty. Aftercare instructions should be time- and action-specific

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VISIT TWO: 14 DAYS LATER

- Chief Complaint: Back Pain
- Vitals: Temp 98.0, pulse 84, respir 16, BP 170/107, pain scale 10/10
- HPI: Persistent low back pain radiating down right leg to knee. No heavy lifting. No additional history but extensive neg. ROS
- Exam: No cervical/thoracic/lumbar tenderness to palpation. Full ROM of back without much difficulty. 5/5 strength bilaterally, 2+ DP / PT pulses bilaterally, normal sensation to light touch bilaterally, normal gait, neg straight leg bilaterally
- Urine negative except for blood
- Diagnosis: Sciatica
- Disposition: Discharged to home. Prescriptions for naproxysn, percocet, prednisone. After care instructions for sciatica.

Risk Management Issues for Return Visit

Whoa baby, talk about diagnosis momentum! Is there *anything* in this history or exam suggesting strain? The

patient is certainly high risk, as this is now a bounceback (though some would call it an annoyance), but with a totally negative exam and no mechanism is still diagnosed with mechanical back pain (sciatica).

Additionally, untreated hypertension is a risk factor for AAA; with the second reading, we do not have a definitive diagnosis, but this is more suggestive of hypertension than one isolated reading.

Visit Three (ED): Two Days Later

- Chief Complaint: Back pain
- Vitals: Temp 98.5 pulse 90 respir 18 BP153/103 pain scale 10/10
- HPI: Persistent right back pain for 7 weeks. Today he has also developed upper abdominal pain and dizziness. Has taken vicodin, Percocet and prednisone with some temporary relief of his symptoms. He denies urinary symptoms, fever, vomiting, chest pain, SOB or headache.
- Exam: ABD: Non-distended, minimal epigastric tenderness-no RUQ tenderness, soft without rigidity, re-

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bound or guarding. Back: Pain with palpation musculature low back. No midline tenderness to palpation. Neuro: Strength 5/5, patellar DTR's normal x2, straight leg raise negative x2, sensation grossly intact bilateral lower extremities. No evidence of urinary incontinence.

- CT: There is a huge 15.6x13.0 cm mass likely representing a renal cell carcinoma of the right kidney with evidence for multiple intrahepatic metastasis and bony metastasis to the lumbar spine.
- Diagnosis: Cancer-urinary system

Discussion: Diagnosis and Management of Acute Low Back Pain

The prevalence of back pain is enormous. Between 70% and 85% of adults will have back pain at some point in their lives; the annual prevalence ranges from 15% to 45%.¹ One of the difficulties in the evaluation of back pain is that it is most often of a benign etiology, and the clinician can be lulled into complacency.

Back pain can be divided into two groups: mechanical/discogenic and non-mechanical.

Mechanical etiologies include idiopathic or nonspecific (strain/sprain) low back pain, discogenic pain, spinal stenosis, and chronic low back pain.

Non-mechanical etiologies include malignancy, infection, inflammation (rheumatologic), gynecologic, renal (urinary tract infection, pyelonephritis, renal colic, renal artery occlusion), gastrointestinal (peptic ulcer disease, pancreatitis), and vascular (ruptured AAA).

Red flags for more serious disease include age >65, history of malignancy, unexplained weight loss, recent trauma, fever, failure to improve after one month of therapy, nocturnal pain, injection drug use, morning stiffness, and history of peripheral vascular disease.²

History and Physical Exam

A directed history should attempt to exclude serious causes of back pain. Inquire about mechanism of injury, onset, and modifying factors, including overthe-counter or other medications which have been tried. Ask about any past history of back pain and the red flags listed previously.

Physical exam includes visual inspection of the back; palpating for vertebral tenderness; percussion for costovertebral angle tenderness; lower extremity strength, sensation and reflexes; and the straight leg test.

Many studies have unsuccessfully attempted to correlate physical exam findings with pain, with the



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Table 1. Representative Results of MRI Studies in Asymptomatic Adults ⁴								
Study	Subjects	Anatomical findings						
		Herniated disk	Bulging disk	Degenerative disk	Stenosis	Annular tear		
				Prevalence (%)				
Boden	Volunteers, <60 yrs old	22	54	46	1	NR		
	Volunteers, ≥60 yrs old	36	79	93	21	NR		
Jensen	Volunteers, mean age 42 yrs	28	52	NR	7	14		
Weishaupt	Volunteers, mean age 35 yrs	40	24	72	NR	33		
Stadnik	Patients referred for head or neck imaging (mean age, 42 yrs)	33	81	72	NR	56		
NR, not reporte	NR, not reported							

available. The sensitivity for plain film is low in most of these conditions (e.g., infection and cancer), so if there is a concern for these entities, MRI should be performed; if this is unavailable in the urgent care center, then the patient should be referred. The main current indication for plain back films is to evaluate for fracture with a traumatic mechanism.

The reflex MRI is controversial, as there are almost no normal results and patient's symptoms are often attributed to incidental findings. The main indication for emergent MRI is for evaluation of an epidural compression syndrome (for example, an abscess, hematoma, or mass causing neurologic symptoms such as urinary retention). **Table 1** illustrates the exceedingly high incidence of abnormal findings in asympto-

exception of straight leg raise testing. The straight leg raise test is performed by using one hand to lift the heel while using the other hand to keep the knee extended. A positive test is the reproduction of sciatica with leg elevation between 30% and 60%. Sciatica is a sharp or burning pain radiating down the posterior or lateral aspect of the leg, often associated with numbness and paresthesia.

Cauda equine syndrome is a rare finding, but worth mentioning since it is one of the true back emergencies. Symptoms include saddle anesthesia (a sensory deficit over the buttocks, posterior superior thighs, and perineal regions), urinary retention, sciatica, sensory and motor deficits, diminished anal sphincter tone and abnormal straight leg raises. Sensitivity of urinary retention is 90% and specificity is 95%.³

Testing

Non-mechanical causes of back pain can be further evaluated if indicated with urinalysis, radiology, and chemistries. Previously, plain films were recommended in those patients who were deemed suitable for further evaluation, such as those with fever, history of cancer, trauma, or weight loss,² but these criteria were established 11 years ago based on earlier data, before MRI was widely matic patients.

Prognosis and management

Between 60% and 70% of patients recover from an acute episode of back pain by six weeks; by 12, weeks 80% to 90% have recovered.

Recovery of patients with herniated disks is no different. Recurrences of pain occur in up to 40% of patients by six weeks.³

Management of acute idiopathic back pain, as well as pain that is caused by a herniated disk, is the same, including non-steroidal anti-inflammatory drugs, muscle relaxants, pain medications, and rapid return to normal activities. Spinal manipulation and physical therapy have a limited effectiveness and should be delayed until pain has persisted for at least three weeks, as 50% of patients will improve in this time.

Therapies shown to be *in*effective include bed rest, back exercises in the acute phase, lumbar supports, facet joint injections, and acupuncture.⁴

Evaluation of the drug-seeking patient

It is well known that back pain is a favorite complaint of the narcotic-seeking patient. Is it possible to separate those patients who have organic disease from those seeking narcotics? In 1980, Waddell devised a set of physical signs to differentiate these patients.⁵ Three or more of the following "Wadell's signs" on exam strongly suggest a non-organic component to back pain:

- 1. Overreaction to the physical exam
- 2. Widespread superficial tenderness that does not correspond to an anatomical distribution
- 3. Pain on axial loading of the skull or simultaneous rotation of the shoulders and pelvis
- 4. Severe limitation on straight leg raise in patients able to sit forward with legs extended
- 5. Weakness or sensory loss that does not correspond to a nerve root distribution

In a small study in 2002, Bloom, et al described the "heel tap" test, which seemed to correlate with Wadell's signs but was easier to perform.

In the heel tap test, the examiner tells the patient that this might cause low back pain, and then gently taps on the patients heels while seated with the hips and knees flexed to 90°. A sudden onset of low back pain is a positive test.⁶

Conclusion

Our patient had no history of back pain and no clear mechanism of injury. His exam was not particularly convincing for a musculoskeletal etiology, as he did not have much pain with range of motion. A CT scan was done to exclude renal stone, and metastatic renal cancer was found.

In the urgent care setting, the first step in back pain evaluation is exclusion of life-threatening or reversible causes—specifically, abdominal aortic aneurysm, epidural compression syndromes, infection, and tumor.

Four screening (ROS) questions for all back pain patients should include:

Continued on page 38

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CLINICAL CHALLENGE CASE 1

In each issue, *JUCM* will challenge your diagnostic acumen with a glimpse of x-rays, electrocardiograms, and photographs of dermatologic conditions that real urgent care patients have presented with. If you would like to submit a case for consideration, please e-mail the relevant materials and presenting information to *editor@jucm.com*.



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INSIGHTS IN IMAGES: CLINICAL CHALLENGE CASE 1

THE RESOLUTION



The x-ray shows a fibular head fracture. The patient was placed in a cast splint across the knee and referred for follow-up with an orthopedist.

This is a very good case for our purpose for what it does *not* show.

Fractures of the proximal fibula are infrequently isolated, and more often associated with significant injury to the surrounding ligamentous structures. Care should be taken to examine the ipsilateral ankle for deformity and tenderness.

A Maisonneuve fracture is an unstable fracture of the medial malleolus of the ankle with disruption of the tibiofibular syndesmosis, and is commonly associated with a proximal fibula fracture.

Acknowledgment: Case presented by Nahum Kovalski, BSc, MDCM, Terem Emergency Medical Centers, Jerusalem, Israel.



CLINICAL CHALLENGE CASE 2



The patient is a 54-year-old woman who presents with a three-day history of constipation. She denies any vomiting, and you note no abdominal distension. She is passing small amounts of gas.

View the image taken (**Figure 1**) and consider what your diagnosis and next steps would be.

Resolution of the case is described on the next page.

INSIGHTS IN IMAGES: CLINICAL CHALLENGE CASE 2

THE RESOLUTION



There are multiple small A/F levels and a small loop of distended small bowel. However, there is gas all the way down to the rectum, which was more evident on the second film (not shown).

In this case, one should consider partial bowel obstruction.

While it may be feasible to try to treat the constipation and then repeat the film eight to 12 hours later to see the progression, it would be more prudent to refer for additional evaluation and treatment.

Acknowledgment: Case presented by Nahum Kovalski, BSc, MDCM, Terem Emergency Medical Centers, Jerusalem, Israel.

These cases are among hundreds that can be found in Terem's online X-ray Teaching File, with more being added daily. Free access to the file is available at https://www2.teremi.com/xrayteach/. A no-cost, brief registration is required.



On Imaging Head Injuries, Routine Cellulitis, Alteplase and Ischemic Stroke, Head Lice, Steroids for Pharyngitis, and Brain Injury in Children

NAHUM KOVALSKI, BSc, MDCM

ach month, Dr. Nahum Kovalski reviews a handful of abstracts from, or relevant to, urgent care practices and practitioners. For the full reports, go to the source cited under each title.

Diagnostic Imaging Rates for Head Injury in the ED and States' Medical Malpractice Tort Reforms

Key point: The authors found a 40% lower incidence of imaging in states with tort reform, compared with states that do not have it in place.

Citation: Smith-Bindman R, McCulloch CE, Ding A, et al. *Am J Emerg Med*. 2010;Jul 12. [Epub ahead of print]

As recently as 2005, studies have found that nearly all physicians reported using defensive medical practices; ordering more diagnostic tests than medically indicated was the most frequently reported practice.

Emergency department physicians are particularly likely to report using defensive medicine. The American College of Emergency Physicians (ACEP) guidelines for neurologic imaging of patients seen in an emergency department for a head injury state that imaging should be used when the injury is severe, but considered discretionary in other cases.

The team used a sample of 8,588 Medicare-eligible women 65 years and older living in 10 U.S. states who presented to an emergency department with head injury between January 1992 and December 2001.

The study team defined the injury as "severe" when the



Nahum Kovalski is an urgent care practitioner and assistant medical director/CIO at Terem Emergency Medical Centers in Jerusalem, Israel. woman had lost consciousness or had presented with an open head wound. They assessed whether CT or MR exams had been performed with seven days of the visit.

Then, the researchers determined whether each state had medical tort reform legislation, classifying laws into four types:

- 1. Caps on monetary damages
- 2. Mandated periodic award payments (these allow losing defendants to pay in installments)
- 3. Collateral source offset rules (which deny compensation for losses that can be recouped from other sources)
- 4. Caps on attorney contingency fees (which limit how much the representing attorney can collect as a percentage of the award)

States with laws that limited monetary damages, mandated periodic award payments, or specified collateral source offset rules had 40% lower odds of imaging, whereas states that had laws that limited an attorney's contingency fees had higher odds of imaging compared to states without these laws.

"[Even] after adjusting for individual and community factors, the total number of laws remained significantly associated with the odds of imaging, and the effect of the individual laws was attenuated but not eliminated," the authors wrote.

The team conceded that the increase in imaging over the decade studied could have been caused by other factors, such as increased availability and accessibility of MR and CT scanners, which may have filled a previously unmet need; patients' increasing assertiveness in requesting imaging; and a decreasing tolerance for uncertainty.

[Published in AuntMinnie.com, August 26, 2010—Kate Madden Yee.

Causes and Treatment of Routine Cellulitis

Key point: Beta-hemolytic streptococci infection was implicated in most cases of nontraumatic cellulitis.

Citation: Jeng A. Beheshti M, Li J, et al. The role of β -hemolytic streptococci in causing diffuse, nonculturable cellulitis: A prospective investigation. *Medicine (Baltimore)*. 2010;89 (4):217-226.

Cellulitis is a diffuse infection that causes redness, heat, and swelling of the skin and underlying soft tissue, particularly on the legs. It should be distinguished from cutaneous inflammation associated with a suppurative focus, such as an abscess, furuncle, or underlying osteomyelitis.

Findings from previous studies that made use of cultures, serology, immunofluorescent staining of skin biopsies for streptococcal antigens, and experimental models in animals have suggested that the vast majority of cases are caused by beta-hemolytic streptococci, not only Group A (*Streptococcus pyogenes*), but other groups as well. Some cases may be caused by *Staphylococcus aureus*, but the role of methicillin-resistant *S aureus* (MRSA) strains has been unclear.

Investigators evaluated 179 patients with cellulitis, excluding those with animal or human bites, foreign bodies, or neutropenia. The patients were tested for acute and convalescent titers of anti-streptolysin O and anti-DNaseB; the former helps to detect infection with streptococcal Groups A, C, and G; the latter detects Group A infections alone.

Along with results from blood cultures, these tests implicated beta-hemolytic streptococci in 73% of cases. In a medical center where MRSA was common in cutaneous abscesses and other skin and soft-tissue infections, 96% of patients receiving beta-lactam antibiotics ineffective against MRSA had a successful outcome.

The excellent response to beta-lactam antibiotics indicates that MRSA is a very uncommon cause of cellulitis. To treat patients with typical nonculturable cellulitis, clinicians can prescribe beta-lactam penicillins, such as parenteral oxacillin or oral dicloxacillin, or first-generation cephalosporins, such as parenteral cefazolin or oral cephalexin.

[Published in *J Watch Dermatol*, August 13, 2010—Jan V. Hirschmann, MD.] ■

Alteplase is Effective Up to 4.5 Hours After Onset of Ischemic Stroke

Key point: Although risk from alteplase was greater when administered at 3 to 4.5 hours, treatment was still beneficial. Citation: Implementation and outcome of thrombolysis with alteplase 3–4.5 h after an acute stroke: An updated analysis from SITS-ISTR. Lancet Neurol. 2010;9:866.

On the basis of reports published in September 2008 from two

large international studies, professional stroke organizations extended the recommended time between symptom onset and administration of alteplase from three to 4.5 hours. To assess implementation of the wider treatment window and its effects, investigators analyzed data for nearly 24,000 patients who were included in one of the study's stroke registry from 2002 to 2010.

Overall, 2,376 patients received alteplase between three and 4.5 hours after symptom onset; the proportion of patients who were treated within this window was three times higher in the last quarter of 2009 than in the first quarter of 2008.

Rates of poor outcomes were low:

7.1% of patients treated within three hours and 7.4% of those treated at three-to-4.5 hours had symptomatic intracerebral hemorrhage

■ 12.3% and 12.0%, respectively, died within three months.

However, in analyses adjusted for confounding variables, patients treated at three to 4.5 hours had significantly higher rates of symptomatic intracerebral hemorrhage (one extra hemorrhage for every 200 patients) and three-month mortality (one extra death for every 333 patients), as well as significantly worse functional outcomes.

Median time from admission to treatment was 65 minutes before and after the reports. The authors conclude that the extended treatment window was implemented rapidly, with no overall increase in admission-to-treatment time, and that although risk from alteplase was greater when administered at three to 4.5 hours, treatment was still beneficial.

Although the U.S. FDA has not yet approved use of alteplase beyond three and up to 4.5 hours after onset of ischemic stroke symptoms, this evidence supports a wider treatment window and professional organizations recommend it.

[Published in *J Watch Emerg Med*, August 27, 2010—Kristi L. Koenig, MD, FACEP.] ■

Head Lice

Key point: Head lice have low contagion in classrooms, and infected children should not be restricted from school attendance. Citation: Frankowski BL, Bocchini JA Jr, Council on School Health and Committee on Infectious Diseases. Head lice. *Pediatrics*. 2010;126(2):392-403.

The American Academy of Pediatrics (AAP) has released a revised clinical report on the management of head lice. It contains a great deal of practical information, including the following highlights:

Background Information

- Lice are common in children aged 3 to 12 years (estimates range from 6 million to 12 million cases per year in the U.S.).
- Empty egg casings or nits are easier to see than viable eggs on darker hair because they are whiter.

ABSTRACTS IN URGENT CARE

- Itching may not develop for four to six weeks after eggs hatch.
- "Lice cannot hop or fly; they crawl."

Diagnosis

- Use of a louse comb facilitates detection of head lice.
- Children should not be sent home from school on the day of diagnosis because they have likely been infected for >1 month and pose little risk to others.
- Children who have had "head-to-head" contact with index cases should be checked.
- Although the intent of a properly worded letter from school is to encourage parents to check their children for lice at home, some experts believe letters cause unnecessary angst among parents.

Policy Recommendations

- Infected children should not be restricted from school attendance. Head lice have low contagion in classrooms.
- The AAP and the National Association of School Nurses discourage a no-nit policy because it is not based on science.
- Head-lice screening programs have not proven effective.

Treatment Recommendations

- Permethrin 1% (Nix) or pyrethrins (Rid, A-200, Pronto) are preferred treatments in communities where resistance has not been reported. Re-treatment nine days after initial therapy is recommended with both products.
- Manual removal of nits immediately after treatment is not necessary.
- Providing parents with instruction in the proper use of any treatment is critical.
- Alternative treatments include:
 - malathion 0.5% (Ovide); for children \geq_2 years
 - benzyl alcohol 5% (Ulesfia); for children ≥6 months
 - permethrin 5% (Elimite); for infants as young as 2 months
 - other treatments that require further evaluation include crotamiton 10% (Eurax), oral ivermectin (Stromectol; for children who weigh ≥15 kg), oral sulfamethoxazole-trimethoprim (Septra), herbal products, occlusive agents (e.g., petrolatum shampoo), and desiccation.

I suspect that many clinicians must "negotiate" with families and schools about the best way to proceed when a child has lice. In addition to this report, the National Pediculosis Association website is an excellent source of information.

[Published in J Watch Pediatr Adolesc Med, August 25, 2010—Howard Bauchner, MD.]

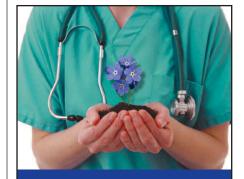
Systemic Steroids for Pharyngitis Pain?

Key point: A meta-analysis suggests modest benefit. Citation: Wing A, Volla-Roel C, Yeh B, et al. Effectiveness of corticosteroid

treatment in acute pharyngitis: A systematic review of the literature. Acad Emerg Med. 2010;17(5):476-483.

A 2009 meta-analysis suggested that administration of corticosteroids for patients with acute pharyngitis increases the likelihood of pain resolution at 24 and 48 hours and hastens pain relief by approximately six hours, particularly in patients with positive bacteriologic tests or presence of exudate.

In the current study, researchers conducted a meta-analysis of the eight



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Come see us at the Fall UCAOA Conference Oct. 21-23 • Booth 2O4, and we'll help you plants the seeds for a successful practice! trials included in the prior analysis plus two additional randomized, controlled trials in which corticosteroids (alone or in combination with antibiotics) were compared with placebo or standard therapy in adults, children, or both.

The 10 studies involved 1,096 patients. Seven studies used dexamethasone (0.6 mg/kg to a maximum of 10 mg orally), and three used prednisone (60 mg orally), betamethasone (2 mL intramuscularly), or cortisone (500 or 600 mg intramuscularly). In pooled analyses, corticosteroids decreased the time to clinically significant pain relief by 4.5 hours. However, at 24 hours, the mean reduction in pain scores associated with corticosteroids (0.9 points on a 10-point visual analog scale) was not clinically significant. No serious adverse events were attributable to corticosteroids.

This study and the prior analysis show a modest improvement in time to pain relief when steroids are added to usual treatment for acute pharyngitis.

Although the data are not compelling, a single oral dose of corticosteroids (e.g., 60 mg of prednisone) is a reasonable option for adults with acute severe pharyngitis with bacterial etiology or exudate.

[Published in J Watch Emerg Med, June 4, 2010—Diane M. Birnbaumer, MD, FACEP.]

Symptoms Following Mild Brain Injury in Children

Key point: Most children will be symptom-free by 1 year. Citation: Barlow KM, Crawford S, Stevenson A, et al. Epidemiology of post-concussion syndrome in pediatric mild trau-

"Bouncebacks" continued from page 29

- 1. fever
- 2. abdominal pain
- 3. weight loss
- 4. urinary retention.

A reasonable initial approach to a patient with low back pain without acute surgical symptoms may be conservative therapy, such as NSAIDS, muscle relaxants, and pain medications. Educate the patient to pursue further evaluation if the pain does not improve within a defined period of time.

Finally, if the mechanism of injury and exam are inconsistent with the diagnosis, an alternate diagnosis should be considered and definite follow-up arranged. The etiology of the patient's pain may not be found on the initial visit, but you can always make sure you follow these golden rules of high-risk patients:

• You first must recognize them.

matic brain injury. Pediatrics. 2010;126(2):e374-e381.

Mild traumatic brain injury (mTBI) occurs in an estimated 692 per 100,000 children younger than 15 years in the U.S. To determine the incidence and natural history of post-concussion symptoms in children with mTBI, researchers at an emergency department in Canada prospectively compared physical, cognitive, emotional, and behavioral symptoms in 670 children with mTBI (age range, 0–18 years) and 197 children with extracranial injury (controls).

The definition of mTBI was admission Glasgow Coma Scale score of 13 to 15, loss of consciousness or altered mental status for <20 minutes, absence of focal neurological deficits, and post-traumatic amnesia for <24 hours.

Parents completed several questionnaires (including a concussion-specific symptom inventory) seven to 10 days after the injury (for pre-injury and current symptom assessment), two weeks later, and then monthly until symptoms resolved.

Pre-injury symptom scores were similar in the two groups. Three months after injury, significantly more children with mTBI than controls were symptomatic (11.0% vs. 0.5%); this significant difference persisted at one year (2.3% vs. 0.01%, respectively).

The most common symptoms at one month were fatigue, more emotional, irritability, and headache.

Age older than 6 years and more-severe mTBI were significantly related to persistence of symptoms.

Parents often ask if their children will have symptoms after mTBI. The vast majority of children will be symptom free by one year.

[Published in J Watch Pediatr Adolesc Med, August 25, 2010— Howard Bauchner, MD.]

- Review your documentation, thoughts, vitals, and any inconsistencies that may be in the history and/or exam.
- Consciously work on a positive relationship with your patients throughout the evaluation, which will not only facilitate communication and enhance the medicine you deliver, but help in risk management issues
- Make sure appropriate and timely follow-up is discussed, documented, and arranged if possible.

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HEALTH LAW

The Checklist—Part 2

JOHN SHUFELDT, MD, JD, MBA, FACEP

(Dr. Shufeldt began a three-part discussion of the importance of procedural checklists in the September issue of JUCM. That column is available at www.jucm.com.)

went to Mardi Gras two years ago. One of the events I attended was called the MOMs Ball. *MOMs* is an acronym for Mystic Orphans and Misfits; it's a party by invite only, and only those with costumes and ticket are admitted.

I was struggling to think of a costume suitable for such a wild event. For example, one guy arrived wearing only a bagel. I will leave it to your imagination how he wore the bagel. Let's just say, a danish would not have worked.

Anyway, since the party was not long after Capt. Chesley "Sully" Sullenberger (aka the Hero on the Hudson) landed an airliner safely in the Hudson River, and since I already had part of the pilot's costume, I decided to go as Sully.

Unfortunately, the blue pilot's blazer at the costume store was not made for someone my size and the pilot's hat did not fit my apparently "walking candy apple-sized cranium." The getup did, however, fit my attorney friend Bill, who was thrilled to wear a hero's costume and receive the subsequent welcome. Regrettably, I was forced to dress as the less-than heroic flight attendant who opened the rear passenger door and flooded the cabin. So, while Bill was getting kissed by a multitude of intoxicated but grateful topless woman, I was fighting off drunken male partygoers bent on groping me so much so that I felt like Jodie Foster in the *Accused*, but I digress.

Unlike my friend "Capt. Bill," Sully did not believe he was a hero. Amid the hoopla surrounding him in the days following the water landing, Sullenberger said, "I want to correct the record right now. This was a crew effort." The outcome had as much to do with his skill as with teamwork, and with their adherence to procedures and checklists.

Climbing through 3,000 feet with copilot Jeff Skiles flying the



John Shufeldt John Shufeldt is principle of Shufeldt Consulting and sits on the Editorial Board of JUCM. He may be contacted at JohnShufeldt@shufeldtconsulting.com. plane, U.S. Airways Flight 1549 crossed through a gaggle of geese, knocking out both engines.

The two aviators' training kicked in immediately. Sullenberger said, "My airplane" and took control. Skiles (who, by the way, also had nearly 20,000 hours of flight time) went right for the checklist. First, he tried to relight both engines, then one engine. Investigators later commented that it was very remarkable that he was able to actually go through these procedures.

He also was working to ensure he went through the most crucial procedures on the ditching checklist. As Skiles methodically went through the checklists, Sullenberger lined up with the Hudson and communicated with air traffic control (ATC):

15:26:54	SULLENBERGER: Flaps up.
15:27:07	SULLENBERGER: After-takeoff checklist com-
	plete.
15:27:10.4	SULLENBERGER: Birds.
15:27:11	SKILES: Whoa.
15:27:	CAM [sound of thump/thud(s) followed by shud-
	dering sound]
15:27:12	SKILES: Oh [expletive deleted].
15:27:13	SULLENBERGER: Oh yeah.
15:27:13	CAM [sound similar to decrease in engine
	noise/frequency begins]
15:27:14	SKILES: Uh-oh.
15:27:15	SULLENBERGER: We got one rol- both of 'em
	rolling back.
15:27:18.5	SULLENBERGER: Ignition, start.
15:27:21.3	SULLENBERGER: I'm starting the APU.
15:27:23.2	SULLENBERGER: My aircraft.
15:27:24	SKILES: Your aircraft.
15:27:25	CAM [sound similar to electrical noise from en-
	gine igniters begins]
15:27:26.5	COMPUTER: Priority left. [Auto callout from the
	computer; this occurs when the sidestick priority
	button is activated on the captain's sidestick.]
15:27:28	CAM [sound similar to electrical noise from en-
	gine igniters ends]
15:27:28	SULLENBERGER: Get the QRH [Quick Refer-
	ence Handbook "checklist"] loss of thrust on both

HEALTH LAW

	engines.
15:27:28	COMPUTER: [sound of single chime begins and
	repeats at approximately 5.7 second intervals
	until]
15:27:32.9	SULLENBERGER: Mayday mayday, mayday. Uh
	this is uh Cactus 15-39 hit birds, we've lost thrust
	(in/on) both engines we're turning back towards LaGuardia.
15.72.42	DEPARTURE: OK, uh, you need to return to La-
15:27:42	Guardia? Turn left heading of uh 2-2-0.
15:27:46	SULLENBERGER: 2-2-0.
15:27:50	SKILES: If fuel remaining, engine mode selector,
19.27.90	ignition. Ignition.
15:27:54	SULLENBERGER: Ignition.
15:27:55	SKILES: Thrust levers confirm idle.
15:27:58	SULLENBERGER: Idle.
15:28:02	SKILES: Airspeed optimum relight 300 knots.
-	We don't have that.
15:28:05	SULLENBERGER: We don't.
15:28:05	DEPARTURE Cactus 15-29, if we can get it for you
	do you want to try to land runway 1-3?
15:28:05	SKILES: If 3-19
15:28:10.6	SULLENBERGER: We're unable. We may end up
	in the Hudson.
15:28:14	SKILES: Emergency electrical poweremergency
	generator not online.
15:28:19	SULLENBERGER: (It's/is) online.
15:28:21	SKILES: ATC notify. Squawk 77 hundred.
15:28:25	SULLENBERGER: Yeah. The left one's coming
	back up a little bit.
15:28:30	SKILES: Distress message, transmit. We did.
15:28:31	DEPARTURE Arright Cactus 15-49 its gonna be
15:28:26	left traffic for runway 3-1.
15:28:36	DEPARTURE OK, what do you need to land? SKILES: (He wants us) to come in and land on 1-
15:28:37	3for whatever.
15:28:45	SKILES: FAC [Flight Augmentation Computer]
13.20.45	one off, then on.
15:28:46	DEPARTURE Cactus 15-(29) runway four's avail-
. 5.20.40	able if you wanna make left traffic to runway four.
15:28:49.9	SULLENBERGER: I'm not sure we can make any
5 15 5	runway. Uh what's over to our right anything in
	New Jersey maybe Teterboro?
15:28:55	DEPARTURE OK yeah, off your right side is Teter-
	boro Airport.
15:29:00	SKILES: No relight after 30 seconds, engine mas-
	ter one and two confirm
15:29:02	DEPARTURE You wanna try and go to Teter-
	boro?
15:29:03	SULLENBERGER: Yes.
15:29:05	SKILES:off.

15:29:07	SULLENBERGER: Off.
15:29:10	SKILES: Wait 30 seconds.
15:29:11	SULLENBERGER: This is the captain brace for im-
	pact.
15:29:14.9	COMPUTER: 1,000 [feet above the ground].
15:29:16	SKILES: Engine master two, back on.
15:29:18	SULLENBERGER: Back on.
15:29:19	SKILES: On.
15:29:21	DEPARTURE Cactus 15-29, turn right 2-8-0, you
	can land runway one at Teterboro.
15:29:21	SKILES: Is that all the power you got? (Wanna)
	number one? Or we got power on number one.
15:29:25	SULLENBERGER: We can't do it.
15:29:26	SULLENBERGER: Go ahead, try number 1.
15:29:27	DEPARTURE OK which runway would you like at
	Teterboro?
15:29:28	SULLENBERGER: We're gonna be in the Hudson.
15:29:33	DEPARTURE I'm sorry say again Cactus?
15:29:36	SKILES: I put it back on.
15:29:37	SULLENBERGER: OK put it back onput it back
	on.
15:29:37	COMPUTER: Too low. Terrain.
15:29:44	SKILES: No relight.
15:29:45.4	SULLENBERGER: OK let's go put the flaps out,
	put the flaps out.
15:29:45	SKILES: Flaps out?
15:29:49	COMPUTER: Terrain terrain. Pull up. Pull up.
15:29:51	DEPARTURE Cactus uh
15:29:53	DEPARTURE Cactus 15-49 radar contact is lost
	you also got Newark airport off your 2 o'clock in
	about seven miles.
15:29:55	COMPUTER: Pull up. Pull up. Pull up. Pull up. Pull
	up. Pull up.
15:30:01	SKILES: Got flaps out.
15:30:03	SKILES: 250 feet in the air.
15:30:04	COMPUTER: Too low. Terrain.
15:30:06	SKILES: 170 knots.
15:30:09	SKILES: Got no power on either one? Try the
15120100	other one.
15:30:09	DEPARTURE: 2-1-0, uh, 47-18. I think he said
15:20:11	he's goin in the Hudson. SULLENBERGER: Try the other one.
15:30:11	-
15:30:14	DEPARTURE Cactus 15-29, uh, you still on? COMPUTER: [Sound of continuous repetitive
15:30:15	chime begins and continues to end of recording.]
	כוווווכ שבצוווז מוום כטוונווומבי נט בוום טו ובכטומוווצ.]
At this poi	nt, the plane was touching down in the water. Sul-
	d pitched the plane's nose up slightly for best glide
	a preside a line plane o hose up singlity for best glide

At this point, the plane was touching down in the water. Sullenberger had pitched the plane's nose up slightly for best glide speed; Skiles lowered the flaps at the last minute to provide further lift at the lower airspeed. After touchdown, Skiles continued to work through the After Ditching Checklist as Sullen-

HEALTH LAW



The author and "Capt. Bill" pay homage to Sully Sullenberger and the crew of U.S Air Flight 1549, Mardi Gras-style.

berger went back to check on the evacuation. You can see while reading the transcript the extreme professionalism and discipline of both Sullenberger and Skiles.

According to Atul Gawande, MD, in *The Checklist Manifesto*, all learned professions have a code of professional conduct:

- Selflessness—Placing the needs of those who depend on us above our own needs.
- Expectation of skill—We always aim for excellence in our knowledge and ability.
- Trustworthiness—We are responsible for our behavior towards those we are in charge of.
- Discipline—The expectation that professionals will follow procedures and work collaboratively and effectively with others. (This is from our aviation brethren.)

One more relevant aside: I am a frustrated rock star. Save for my lack of any musical talent (I have lost friends after singing *Happy Birthday* to them), I could be David Lee Roth.

I recently went to see Van Halen, a band which Roth once fronted. You may remember him. He was the nut-job who insisted on having a large bowl of M&Ms provided to him backstage before concerts. He had one clause in his contract which stated that no brown M&Ms were allowed in the bowl. If any brown M&Ms were found in the bowl, he had the unilateral right to cancel the show with full compensation for the band. In fact, he canceled a show in Colorado after finding a single brown M&M.

As Roth explained in his book *Crazy from the Heat*, Van Halen was one of the first groups to play in the large-scale stadium shows. They would arrive with 10 large semi-trailer trucks full of equipment.

His contract read like the yellow pages. Deep in the contract, in Clause 126, was the "no brown M&M" sentence. Roth said if he saw one brown M&M in the bowl, he knew that other items would be missed as well.

Sure enough, in Colorado, the promoter had not read the weight requirement for the stage and the song *Jump* would have turned into *Fall* as the entire staging would have plunged through the arena floor. Even nut-job David Lee Roth used a preconcert checklist.

Why then, if they are good enough for pilots and big-hair 80s rock bands, has the medical profession been one of the last to embrace the use of

checklists? Historically, the medical profession valued autonomy which is in direct contradistinction to discipline. In medicine today, to overcome necessary fallibility (see last month's column), success now depends on a team of individuals working in concert to provide the best care for the patient.

Over the years, I have introduced a large number of checklists and standing orders. Here are some of the resultant comments from providers:

- "I did not go to medical school to be told how to practice."
- "This is cookbook medicine."
- "These are idiotic, everyone knows this already."
- "The computer told me what to do."
- "These standing orders are for morons; I already know all this."

A multitude of studies have been done in hospitals around the world, showing that the use of these "idiotic checklists" saves lives, prevents infections, alerts the team to potential etiologies for diseases, prevents wrong-side surgery, etc., etc.

Despite our natural inclination toward autonomy and independent thinking, it is clear that the disciplined use of checklists in medicine has come of age.

The final installment of this series next month will discuss how to write and implement useful checklists for an urgent care practice.



OCCUPATIONAL MEDICINE

Just Say 'No' to Cold Calls

FRANK H. LEONE, MBA, MPH

n sales, the term "cold call" may mean different things to different people. I define a cold call as an unannounced visit to a prospect company, whether the intention is to seek an unscheduled meeting or to drop off literature and/or gifts.

I do not consider an initial telephone call a cold call, presuming it follows an introductory letter and/or email correspondence advising the prospect of the impending call.

Consider the following, and the negatives associated with unexpected drop-ins:

- Sometimes cold calls do, in fact, work. Put me in a major league baseball uniform and I might get a hit or two just by swinging. The same is true with cold calls; they work just often enough to keep you coming back.
- 2. It's a heck of a way to make a first impression. Do you have a well-meaning neighbor or friend who frequently knocks on your door unannounced? It's OK sometimes, but a bit annoying when you are engrossed in another matter.

So it is with cold-calling; as often as not, the prospect not only spurns the sales advance but also finds it annoying. Sales is about developing relationships, not dooming them from the start.

3. A face-to-face cold call is not necessarily a victory. Even if you do get in the door for an impromptu meeting, that meeting might not occur within the prospect's time comfort zone.

Every in-person meeting should be dictated by the prospect's schedule, not the sales professional's schedule.

4. Don't toss preparation to the wind. Reviewing a prospect's website prior to a cold call is not appropriate preparation.

Frank Leone is president and CEO of RYAN Associates and executive director of the National Association of Occupational Health Professionals. Mr. Leone is the author of numerous sales and marketing texts and periodicals, and has considerable experience training medical professionals on sales and marketing techniques. E-mail him at *fleone@naohp.com*. When you use the real sales cycle (letter—phone call—reminder—appointment—follow-up), you are increasing the odds of walking in better prepared. For example, a scheduled in-person call can be preceded 24 hours in advance by a call from clinic management to set the stage for the sales professional's call. You can't create such an edge during a cold call.

- 5. Time is money. A sales professional's most valuable commodity is time; an hour saved is an hour earned for more targeted and planned sales calls. Time spent doing one or more personal cold calls involves round-trip auto travel, parking, and waiting time. Two hours of unproductive cold-call time can easily be converted to two hours of active telephone time from the comfort of your office.
- 6. Forget the "leave them some literature" myth. Even in conjunction with an unsuccessful sales call, you have the opportunity to leave literature behind for the prospect to review. Thus, the thinking goes, even if you don't get a face-to-face meeting, you can connect with the prospect by leaving something behind.

Literature or brochures, however, are invariably discarded. If someone left behind literature at my office, for example, I would consider it a negative. It would strike me as an impersonal, even desperate move, and I would be less, not more, inclined to welcome overtures in the future.

At times, prospects actually suggest that they would prefer to receive literature from a program as a first step. However, this approach is likely to be counter-productive. As the saying goes, "Watch what they do, not what they say." The odds are very high that said literature will never be reviewed and that such a request is but a way to defer the sales professional.

Are there times and/or circumstances when a cold call can be justified? Sure. When it comes to sales and marketing, nothing is set in concrete.

Continued on page 44



CODING Q&A

ICD-9 Updates for 2011

DAVID STERN, MD, CPC

pdates to the ICD-9 code set went into effect October 1, 2010. There will be one more regularly scheduled ICD-9 update on October 1, 2011. No update is scheduled for 2012, but on October 1, 2013, the vastly larger ICD-10 code set is scheduled to take effect. The following are changes that are of particular interest to us in the urgent care field:

- New code to specify post-traumatic seizures: When a patient experiences seizure(s) as a result of a head injury, physicians now can use a specific code for this condition— ICD-9 code **780.33**, Post-traumatic seizures.
- New code for jaw pain: Physicians now have code 784.92 to specify the symptom of jaw pain. (In the past, we had to code jaw pain with the nonspecific code 526.9, Unspecified disease of the jaws, which is still a valid code.)
- New codes related to influenza: To specify pneumonia and other manifestations of avian or novel H1N1 influenza, physicians should use the now-expanded codes 488.01– 488.09 and 488.11–488.19, as shown in Table 1.
- Codes related to retained foreign bodies: Physicians can now indicate the presence of retained foreign bodies with new codes of the V90 series, which classify the retained foreign body by its material composition (Table 2).

When appropriate, two other specific codes should be used in addition to the above codes to indicate the anatomic location of the foreign body:

- 360.61, Foreign body in anterior chamber
- **729.6**, Residual foreign body in soft tissue
- New codes related to intrauterine contraceptive devices (IUDs): Use the following new ICD-9 codes for visits related to insertion and removal of IUDs:



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Table 1. New Codes Related to Influenza	
ICD-9 Code	Description of Influenza Condition
488.01	Influenza due to identified avian influenza virus with pneumonia
488.02	Influenza due to identified avian influenza virus with other respiratory manifestations
488.09	Influenza due to identified avian influenza virus with other manifestations
488.11	Influenza due to identified novel H1N1 influenza virus with pneumonia
488.12	Influenza due to identified novel H1N1 influenza virus with other respiratory manifestations
488.19	Influenza due to identified novel H1N1 influenza virus with other manifestations

Table 2. New	Table 2. New Codes Related to Retained Foreign Bodies	
ICD-9 Code	Composition of Retained Foreign Body	
V90.01	Retained depleted uranium fragments	
V90.09	Other retained radioactive fragments	
V90.10	Retained metal fragments, unspecified	
V90.11	Retained magnetic metal fragments	
V90.12	Retained nonmagnetic metal fragments	
V90.2	Retained plastic fragments	
V90.31	Retained animal quills or spines	
V90.32	Retained tooth	
V90.33	Retained wood fragments	
V90.39	Other retained organic fragments	
V90.81	Retained glass fragments	
V90.83	Retained stone or crystalline fragments	
V90.89	Other specified retained foreign body	
V90.9	Retained foreign body, unspecified material	

- **V25.11**, Encounter for *insertion* of intrauterine contraceptive device
- **V25.12**, Encounter for *removal* of intrauterine contraceptive device
- V25.13, Encounter for *removal and reinsertion* of intrauterine contraceptive device

CODING Q&A

Table 3. New Codes Related to Cognitive Abnormalities	
ICD-9 Code	Description of Cognitive Abnormalities
799.51	Attention or concentration deficit
799.52	Cognitive communication deficit
799.53	Visuospatial deficit
799.54	Psychomotor deficit
799.55	Frontal lobe and executive function deficit
799-59	Other signs and symptoms involving cognition

Table 4. New Codes Related to Disorders of Defecation	
ICD-9 Code	Defecation Disorder
787.60	Full incontinence of feces
787.61	Incomplete defecation
787.62	Fecal smearing
787.63	Fecal urgency
560.32	Fecal impaction

Table 5. New Codes Related to Obesity	
ICD-9 Code	Degree of Morbid Obesity
V85.41	Body Mass Index 40.0-44.9, adult
V85.42	Body Mass Index 45.0-49.9, adult
V85.43	Body Mass Index 50.0-59.9, adult
V85.44	Body Mass Index 60.0-69.9, adult
V85.45	Body Mass Index 70 and over, adult

- The code **V25.1** for insertion of an intrauterine contraceptive device is now *invalid*.
- Continue reporting code **V25.42** for routine checking of intrauterine contraceptive device.

OCCUPATIONAL MEDICINE

There are marked downsides to engaging in such exceptions, however.

Think of a recovering alcoholic; he might take a drink at some point, thinking, "What's one drink?" But that drink is likely to lead to a "just one more can't hurt" mentality and, in short order, the alcoholic falls off the wagon.

In sales, one exception leads to two, then three, and soon to a return to the bad old habit of sequential cold calls. I met recently with an experienced sales professional whose sales plan overwhelmingly centered around cold

- New codes for cognitive abnormalities: The new codes detailed in Table 3 allow physicians to better specify types of cognitive deficits.
- New codes for lumbar spinal stenosis: Physicians can now use different codes for spinal stenosis to differentiate between patients with and those without neurogenic claudication:
 - **724.02** Spinal stenosis, lumbar region, without neurogenic claudication
 - **724.03** Spinal stenosis, lumbar region, with neurogenic claudication
- New code for disorders of defecation: Codes in the 787.6 series have been added to specify disorders of defecation (Table 4). Code 560.39 (Other impaction of intestine) is still valid, but should no longer be used to code for a diagnosis of fecal impaction.
- Codes related to Obesity. New V85.4 series (Table 5) codes allow physicians to classify levels of morbid obesity by body mass index.

A related new code (**278.03**) indicates the diagnosis of obesity hypoventilation syndrome.

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calls. When I raised my concern, she countered by telling me that she had worked her comparatively small market for 11 years with a previous employer, and knew most of the key contacts in her community. If there were ever an exception to the in-person, no-cold-call rule, this was it. Yet, all in all, I believe she could have leveraged her name and personal relationships just as effectively from her office.

Respect your prospect's time, above all. Cold calls fall short in this respect. \blacksquare

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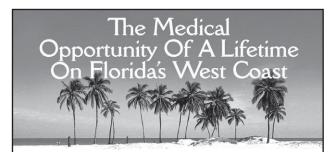
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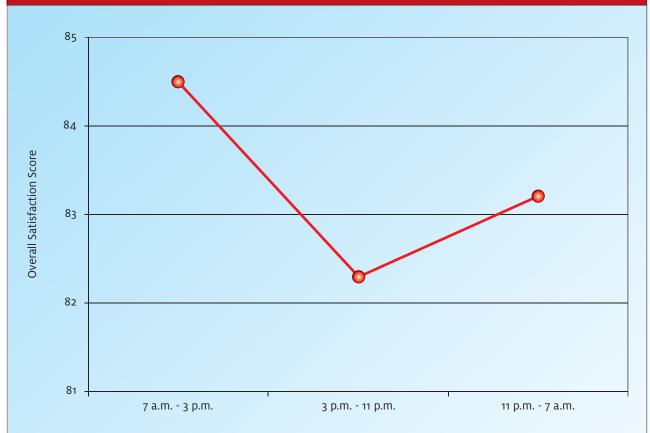
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DEVELOPING DATA

n each issue on this page, we report on research from or relevant to the emerging urgent care marketplace. This month, we relay data that track patient satisfaction with the emergency room based on time of day respondents arrived for treatment.





Source: Pulse Report 2009: Emergency Department. Patient Perspectives on American Health Care and Emergency Department Pulse Report 2007. Patient Perspectives on American Health Care. Press Ganey Associates, Inc.

While these data do not take into account wait times, per se, the authors note that "Staffing patterns, patient volume, and acuity of patient conditions may play a large part in these differences in satisfaction. By mid-afternoon, wait times may be on the rise as patient volumes have increased during the day."

Presumably, at least some of the dissatisfied patients who presented to the ED could have been treated successfully in the urgent care setting.

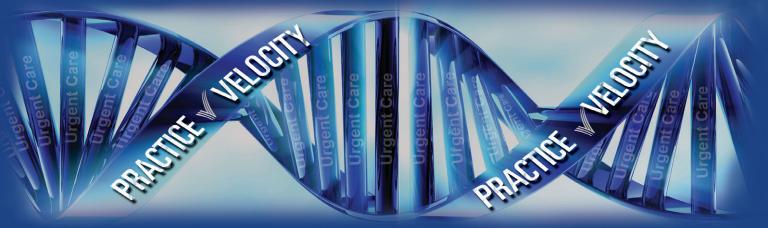
Would such patients in your area know where to find you, and what your hours are? And have you considered forging a referral relationship with nearby hospitals to handle overflow from their ED?

If you are aware of new data that you've found useful in your practice, let us know via e-mail to *editor@jucm.com*. We'll share your discovery with your colleagues in an upcoming issue of *JUCM*.



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