MINOR EMERGENCIES 4e

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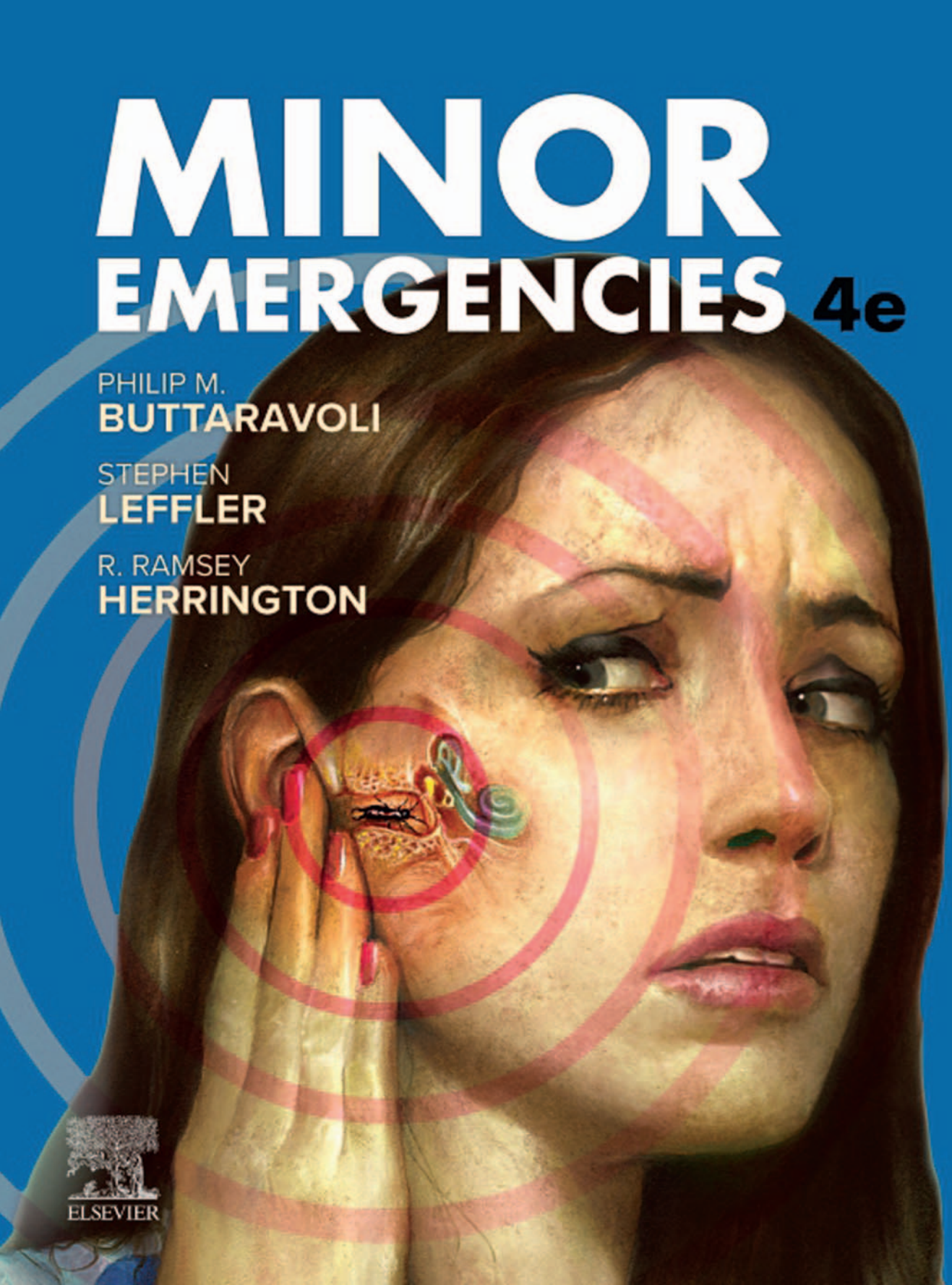

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MINOR EMERGENCIES

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To my special partner Jennifer Stanley, an inspiration as to what vision, dedication, and perseverance, along with bottomless energy, can accomplish. She has supported me in so many ways throughout the writing of this fourth edition. I will be forever grateful.

—Philip M. Buttaravoli

To my wife Robyn, thank you for your ongoing support no matter what life brings our way. To Philip Buttaravoli, thank you for inviting me to be a part of the third and now fourth editions of *Minor Emergencies*.

—Stephen Leffler

To Drs. Phil Buttaravoli, Ruth Uphold, Steve Leffler, Ray Keller, Dave Clauss, and Peter Weimersheimer, the physician leaders in Emergency Medicine who blazed the trail of academic growth at the University of Vermont while fulfilling our clinical mission.

—R. Ramsey Herrington

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Foreword

Foreword to *Minor Emergencies, 4th Edition*

I could not be happier to write an introduction to the textbook *Minor Emergencies*. Diseases described here occur commonly, and most of the clinical situations presented in this book will present daily. This is the material that forms the body of an emergency medical practice. Treatment of these illnesses and injuries is the basis of an acute care practice. These are the complaints from which, if handled well, most patients recover.

During my emergency medical career, I opened the chest and crossed-clamped aortas at least 12 or 13 times. All stopped bleeding, but none of the patients lived longer than 12 hours. This is not the book for this problem. Every patient presenting with a corneal foreign body that I treated (and there were probably more than a thousand) got better. Simple, straightforward problems often can have excellent outcomes. Simple does not mean unimportant, however. Just ask the corneal foreign body patient how they felt just before and just after the topical pain medication.

In emergency medicine, as in theater acting, there are no small parts, and people who think there are do not understand the significance of their role. Patients deserve to have each complaint to be properly evaluated and treated. Proper history, correct physical examination, evaluation, and treatment are still the basis of care. To the patient and their family members doing it right always matters. To the patient's mind, there is no such thing as a minor emergency—if it is me or my family involved, I expect it to be taken seriously.

This textbook reminds us that no matter what the patient's complaint may be, calming the patient, proper evaluation, and direct approach are always best. Each medical problem discussed in this book has both psychological and physical dimensions, and both need to be addressed. The days of the omnipotent doctor are gone. Involving the patient in their care is the therapy of choice. To the patient, everything is new and frightening. Making them a partner in their care is the best technique we can use.

Gregory L. Henry, MD, FACEP
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Preface

Preface to the Second Edition

"Good judgment comes from experience, and a lot of that comes from bad judgment."

—Will Rogers

As a medical student at the University of Vermont in the late 1960s interested in emergency room care (this was considered peculiar at the time), I found myself disappointed that my medical education (excellent in every other way) was lacking when it came to the treatment of simple minor emergencies. I had this in mind when, in 1975, as the medical director of the emergency service at George Washington University Medical Center (and the first residency-trained emergency physician in the Washington, DC, area), I was given the opportunity to present a 1-hour lecture to their medical students on emergency medical care—"Common Simple Emergencies." (At that time, 1 hour was considered very generous for covering all of emergency medicine.)

I eventually expanded this slide show and lecture to a 6-hour series, which I presented regularly at the Georgetown University Medical Center Emergency Department. Even though there were still few published data on most of the topics covered in the lecture series, in 1985, with the help of emergency medicine attending physician Dr. Thomas Stair, I turned "Common Simple Emergencies" into a 300-page book. For the most part, the information contained within this publication was based on common practice and personal experience.

Fifteen years later, with more published data available, the book was again published under the present title and was expanded to 500 pages. The general format ("What To Do/What Not To Do") was maintained. Even with the greater volume of information, the book remained a practical guide.

Today, in stark contrast to when the original edition was published in 1985, there is a plethora of scientific data on most of the subjects covered in *Minor Emergencies*. The book has now grown to over 800 pages. In the face of the sometimes overwhelming volume of data now available, I have endeavored to continue to present these topics on minor emergencies in a manner that will still allow this larger text to be a useful and practical guide.

I have maintained the simple basic format used in the previous edition and have continued to use bold font to bring the reader's eye to the key information in each chapter. I have added red font to help identify different topics within the text. The discussions are now highlighted and compressed using small font and double columns. These changes have allowed me to make the book more complete and comprehensive, yet still allow it to remain useful at a glance.

The clinical material has all been updated, new topics have been added, and I have used evidence-based data whenever available. Many more photographs and drawings have been added (in color) to benefit the reader. In addition, I have personally reviewed the index to help ensure its usefulness and have attempted to include many identifying symptoms in the index to help users find the topic they are searching for.

I have done all of this so that you as a clinician can have more fun with your patients. When emergencies are minor, it gives you an opportunity to lighten up and enjoy the art of healing. Patients appreciate a confident clinician with a good sense of humor who can stop the pain and/or the worry, fix the problem in a compassionate way, and make them laugh through the process. This book can provide you with the information that you need to perform competently and to relax when presented with the minor emergencies that patients will always need your help with. (You will have to supply the humor.) You will be greatly rewarded for your treatment by seeing their smiling faces and hearing their expressions of gratitude after happily making them well.

Philip M. Buttaravoli, MD, FACEP

Preface to the Third Edition

To incorporate an academic element to the latest edition of *Minor Emergencies*, I have returned to my alma mater, the University of Vermont, thereby bringing the book full circle to its earliest origins. I asked Emergency Department Medical Director Stephen Leffler MD, FACEP whether he and the rest of the emergency department medical staff would be interested in updating the clinical material in *Minor Emergencies* and bringing the book into the digital age with an electronic publication that would include video displays.

Steve, along with his department staff, accepted the challenge enthusiastically.

With their involvement, this latest edition of *Minor Emergencies* should prove to be more accurate and convenient for the user. There will be periodic updates of the electronic version, and this will maintain a continuous renewal of clinical information.

The book title of this third edition has been shortened with the elimination of the subtitle "Splinters to Fractures." This subtitle was thought to be more misleading than informative, and the new abbreviated title better reflects the book's true essence.

It is my hope that this new edition will continue to provide support for all clinicians out there who are caring for the public's minor emergencies on a daily basis.

Philip M. Buttaravoli, MD, FACEP

A Note From the Authors

We have no relationships with or financial interests in any commercial companies that pertain to any of the products mentioned in this publication.

Any comments, suggestions, and/or questions can be directed to Drs. Buttaravoli and Leffler at e-med@juno.com and Stephen.Leffler@vtmednet.org under the subject heading Minor Emergencies.

Philip M. Buttaravoli, MD, FACEP
(Butter ah'voli)
Stephen Leffler, MD, FACEP

Preface to the Fourth Edition

After having won first place in the surgical division of the British Medical Association book awards, we believe there is no reason to change the basic formula for success that we created in the third edition. Over time, though, it does become necessary to inform our readers of the new developments in the field of minor emergencies.

To bring the fourth edition up to date, Dr. Leffler and I have brought Dr. Ramsey Herrington on board as an additional lead author. Dr. Herrington is the chairman of the Department of Emergency Medicine at the University of Vermont Medical Center, where he has been instrumental in establishing a new Emergency Medicine Residency Program.

The emergency physician members of this new program have been key participants in contributing to the update of the fourth edition's new and established chapters. The basic format has stayed the same, and the book should remain a practical guide for both seasoned practitioners and those health care workers who are just starting their careers in emergency medicine, urgent care, and/or family practice.

This will be the last time that I will be actively involved with the publication of *Minor Emergencies*, and I can only hope that I have helped to fill in a small niche that was initially missing from our medical education. I thank everyone who has seen the value in treating minor emergencies with the respect that they deserve.

Philip M. Buttaravoli, MD, FACEP

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Acknowledgments

We have enjoyed the special opportunity to work with Nani Clansey, who has always been a delight to work with and has been most informative and supportive in the creation of this fourth edition. In addition, it has been a pleasure working with the extremely competent, efficient, and hardworking assistance of Charlotta Kryhl, Commissioning Editor, and Karthikeyan Murthy, Project Manager, at Elsevier.

Also, special thanks to the contributing emergency department medical staff at the University of Vermont who will be taking over full responsibility for updating future editions of this publication.

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Video contents

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PART 1

Neurologic and Psychiatric Emergencies

Evie Marcolini ■ Matthew S. Siket

CHAPTER 1

Acute Dystonic Drug Reaction

Presentation

The patient with a dystonic reaction to a neuroleptic or other agent typically presents to the emergency department (ED) or urgent care center with posturing, facial grimacing and involuntary muscle movements, and/or difficulty speaking. Pain is minimal, if at all. The jaw, tongue, lip, throat, and neck muscles are frequently involved. Hyperextension and lateral deviation of the neck along with upward gaze is a classic presentation (Fig. 1.1). Often no history is available. The patient may not be able to speak, may not be aware of taking any phenothiazines or butyrophenones (e.g., Haldol that has been used to cut heroin), may not admit to using an illicit drug or psychotropic medication, or may not make the connection between the symptoms and drug use (e.g., one dose of Compazine given to treat nausea or vomiting). The drugs that are most likely to produce a classic dystonic reaction are prochlorperazine (Compazine), haloperidol (Haldol), chlorpromazine (Thorazine), promethazine (Phenergan), and metoclopramide (Reglan), but the list is long and includes some common agents such as benzodiazepines and antihistamines. Acute dystonia usually presents with one or more of the following types of symptoms:

Buccolingual—protruding or pulling sensation of the tongue

Torticollis—twisted neck or facial muscle spasm

Oculogyric—roving or deviated gaze

Tortipelvis—abdominal rigidity and pain

Opisthotonic—severe hyperextension of entire spinal column

Acute dystonia can resemble partial seizures, the posturing of psychosis, or the spasms of tetanus, strychnine poisoning, or electrolyte imbalance.

More chronic neurologic side effects of phenothiazines, including the restlessness of akathisia, tardive dyskinesia, and parkinsonism, do not usually respond as dramatically to drug treatment as does acute dystonia. Onset of oculogyric crisis and torticollis reactions usually

Fig. 1.1 Patient with dystonic drug reaction.

occurs within a few minutes or hours but may occur 12 to 24 hours after treatment with a high-potency neuroleptic such as haloperidol.

What to Do

Maintain a high degree of suspicion for involvement of an offending drug even when the patient does not volunteer such an exposure. Do the required detective work to reveal any chance of the intake of one of the suspected drugs.

When such a suspicion is confirmed:

- **Administer 1 to 2 mg of benztropine (Cogentin) intravenously (IV)/intramuscularly (IM) or 25 to 50 mg of diphenhydramine (Benadryl) IV/IM,** and watch for improvement of the dystonia over the next 5 minutes. Usually the medication begins to work within 2 minutes of IV administration, and the symptoms completely resolve within 15 minutes. **This step is both therapeutic and diagnostic.** Benztropine produces fewer side effects (mostly drowsiness) and may be slightly more effective, but diphenhydramine is more likely to be available in the ED or urgent care center.

Benztropine may be given to children older than 3 years of age at the dose of 0.02 to 0.05 mg/kg IV, IM, or orally. Diphenhydramine may be given to pediatric patients age 2 to 11 years at 1 to 2 mg/kg IM/IV every 6 to 8 hours as needed, max dosing 50 mg/dose and up to 300 mg/day; orally for patients less than 6 years old is not recommended; 6 to 12 years, 25 mg or 5 to 10 mL orally every 4 to 6 hours.

Instruct the patient to discontinue use of the offending drug and arrange for follow-up if medications must be adjusted. **If the culprit is long acting, prescribe benztropine 1 to 2 mg orally twice a day × 2 to 3 days for adults, or diphenhydramine 25 mg orally every 6 hours for 24 to 72 hours to prevent a relapse.**

Pediatric patients should be admitted for monitoring if a long-acting agent has been ingested.

What Not to Do

Do not immediately begin a comprehensive diagnostic workup. It may not be necessary. If findings are typical, administer benztropine or diphenhydramine first to see if symptoms completely resolve.

Do not confuse dystonia with tetanus, seizures, stroke, psychosis, meningitis, or dislocation of the mandible. None of these will resolve with IV benztropine or diphenhydramine.

Do not persist with treatment if the response is questionable or there is no response. Continue with the workup to find another cause for the dystonia (e.g., tetanus, seizures, hypomagnesemia, hypocalcemia, alkalosis, neuromuscular disease).

Do not use a benzodiazepine to relieve the dystonia. Recognize that a benzodiazepine may resolve spasms from many agents, but it will not help to diagnose a neuroleptic agent as the cause.

Discussion

Dystonic reactions have been reported in 10% to 60% of patients treated with a neuroleptic medication, most commonly when patients just start or increase the dose of the drug or the drug is administered too rapidly through the IV route. Patients with a family history of dystonia, patients with recent use of cocaine or alcohol, younger patients, male patients, and patients already being treated with agents such as fluphenazine or haloperidol are at higher risk for a dystonic reaction.

Dystonia is idiosyncratic, not the result of a drug overdose. The extrapyramidal motor system depends on excitatory cholinergic and inhibitory dopaminergic neurotransmitters, the latter being susceptible to blockage by phenothiazine and butyrophenone medications. Anticholinergic medications restore the excitatory–inhibitory balance. **One IV dose of benztropine or diphenhydramine is relatively innocuous, rapidly diagnostic, and probably justified to be used as an initial step in the treatment of any patient with a dystonic reaction.** IM administration may take as long as 30 minutes before an effect is seen.

Suggested Readings

Jhee, S. S. (2003). Delayed onset of oculogyric crisis and torticollis with intramuscular haloperidol. *Annals of Pharmacotherapy*, *37*, 1434–1437.

Lee, A. S. (1979). Treatment of drug-induced dystonic reactions. *Journal of the American College of Emergency Physicians*, *8*, 453–457.

Marano, M., di Biase, L., Salomone, G., et al. (2016). The clinical course of a drug-induced acute dystonic reaction in the emergency room. *Tremor and Other Hyperkinetic Movements*, *6*, 436. PMID 28105387.

CHAPTER 2

Heat Illness

(Heat Syncope, Heat Cramps, Heat Exhaustion)

Presentation

Heat illnesses comprise a spectrum of illnesses resulting from failure of the body's normal thermoregulatory mechanisms after exposure to excessive heat. Most heat-related illness is mild; however, severe hyperthermia associated with heat stroke, neuroleptic malignant syndrome, or serotonin syndrome is a severe, life-threatening condition and should not be overlooked.

The milder forms of heat-related illness include heat syncope (or presyncope), or heat cramps. These illnesses are usually found after prolonged exposure to excessive heat and humidity in patients who are unable to remove themselves from the situation.

Heat syncope is postural syncope or presyncope related to excessive heat exposure.

Heat cramps are painful muscle cramps after vigorous exertion in hot environments (often several hours later) in the calves, thighs, and/or shoulders.

Heat exhaustion is a slightly more severe form of heat illness, but is easily treated with hydration and cooling. Elderly patients (without air-conditioning on a hot, humid day), workers, or athletes (exerting themselves in a hot climate while taking in an inadequate amount of fluid) may be more symptomatic, with fatigue, weakness, lightheadedness, headache, nausea, and vomiting in addition to orthostatic symptoms and painful muscle spasms. **The patient may have a normal temperature (but generally >38 °C), or the temperature may be elevated to 40 °C (104 °F), with tachycardia, clinical evidence of dehydration, and (often, especially with exertion) profuse sweating. Mental status is normal.**

Another minor form of heat stress is heat edema, which occurs in elderly individuals and consists of swelling of the feet and ankles in response to extreme heat. Miliaria, also known as heat rash or prickly heat, is common in hot and humid climates and presents with small, red, pruritic papules that result from plugging of sweat gland ducts.

The severe forms of heat-related illness, such as heat stroke, are characterized by alteration in mental status associated with hyperthermia (temperature >40.5 °C). Neuroleptic malignant syndrome and serotonin syndrome are not typically classified as heat-related illnesses but present with severe hyperthermia and altered mental status and can be easily confused with heat stroke.

What to Do

Assess and monitor all patients with minor heat illness for the development of heat stroke. This is a much more serious form of heat illness, **accompanied by a core temperature**

of greater than 40 °C and altered mental status that can lead to delirium, seizures, or coma.

Remove patients with any form of heat illness from the hot environment. Clothing should be removed to promote cooling, and a temperature obtained (rectally, if possible).

Obtain a careful history from the patient or witnesses, with special attention to the type and length of heat exposure, recent hydration and nutrition, any underlying medical problems, and any medications being used that might predispose the patient to developing heat illness.

Perform a physical examination, noting abnormal vital signs, signs of associated medical illness, evidence of dehydration, and/or diaphoresis.

For heat syncope or presyncope, remove the patient from the source of heat, allow patient to rest, and administer oral or intravenous rehydration. Evaluate for any injury resulting from a fall, and **all potentially serious causes of syncope should be considered** (see Chapter 11).

For isolated heat cramps, provide muscle stretching and massage, and administer an oral electrolyte solution (0.5 tsp table salt in 1 quart of water) or intravenous normal saline for rapid relief.

For heat exhaustion, provide intravenous rehydration with normal saline or a glucose-in-hypotonic saline solution, such as D5 in .45% sodium chloride (1 L over 30 minutes). Obtain serum sodium, potassium, glucose, magnesium, calcium, and phosphorus levels, as well as hematocrit, blood urea nitrogen, and creatinine levels. Correct electrolyte abnormalities appropriately. Avoid rapid correction of hypernatremia, as this can cause cerebral edema.

With temperature above 40 °C, and normal mental status, spray or sponge the patient with tepid or warm water (to prevent shivering) and then fan to enhance evaporation and cooling. Refrigerated gel packs or ice packs may be applied to the forehead, neck, axillae, and groin. Ice water immersion is most effective for rapid cooling but poorly tolerated in most patients (especially elderly patients).

If not treated properly, heat exhaustion may evolve to heatstroke, a major medical emergency that may lead to cardiac arrhythmias, rhabdomyolysis, serum chemistry abnormalities, disseminated intravascular coagulation, irreversible shock, and death. Core or rectal temperature monitoring, physical examination, and laboratory analysis should provide the correct diagnosis.

When a mild form of heat illness responds successfully to treatment, with vital signs returning to normal and symptoms relieved, the patient may be discharged with instructions on how to avoid future episodes and advised to continue adequate fluid intake over the next 24 to 48 hours. Elderly and mentally ill patients and their caregivers should be encouraged to maintain adequate fluid intake to prevent recurrence. Those who must work in a hot environment with high humidity should be encouraged to acclimate themselves over several weeks. Successive increments in the level of work performed in a hot environment result in adaptations that eventually allow a person to work safely at levels of heat that were previously intolerable or life threatening.

Elderly patients and their caretakers, as well as parents of small children, should be educated about high-risk situations and instructed about putting limits on activity during hot and humid days.

Admission should be considered for any patient who presents with altered mental status, heat stroke, or altered electrolytes and elderly patients who have chronic medical problems, significant electrolyte abnormalities, or risk for recurrence. All patients who are treated but do not have a complete resolution of their symptoms over several hours should also be admitted.

What Not to Do

Do not do a comprehensive laboratory workup on young, healthy patients with minimal symptoms or minor heat-related illness.

Do not use pharmacologic agents that are designed to accelerate cooling. None have been found to be helpful. The role of antipyretic agents in heat illness has not been evaluated.

Do not continue therapeutic cooling techniques after the temperature reaches 38.5 °C. Beyond this point, continued active cooling may result in hypothermia.

Do not recommend salt tablets to prevent heat illness. Fluid losses during exercise are much greater than electrolyte losses.

Do not overlook the possibility of neuroleptic malignant syndrome and serotonin syndrome with patients who have recently begun taking neuroleptic drugs or serotonergic agents.

Discussion

Hyperthermia, defined as a core temperature above 40 °C, may present with sweating, flushing, tachycardia, fatigue, lightheadedness, headache, and paresthesia, progressing to weakness, muscle cramps, oliguria, nausea, agitation, hypotension, syncope, confusion, delirium, seizures, and coma. Mental status changes and core temperature distinguish potentially fatal heat stroke from heat exhaustion.

Control of thermoregulation resides within the hypothalamus, which stimulates cutaneous vasodilation and sweating through the autonomic nervous system in response to elevation of blood temperature. Blood flow to the skin may increase 20-fold. Cooling normally occurs by transfer of heat from the skin by radiation, convection, and evaporation. As the ambient temperature exceeds the body's temperature, a rise in body temperature may occur in response to radiation and convection of heat from the environment. When the humidity rises, the body's ability to cool through evaporation is diminished.

Dehydration and salt depletion impair thermoregulation by reducing the body's ability to increase cardiac output needed to shunt heated blood from the core circulation to the dilated peripheral circulation. Cardiovascular disease and use of medications that impair cardiac function can also result in increased susceptibility to heat illness.

Although athletes are commonly thought to be most at risk for heat illness, children and the elderly, poor, and socially isolated are particularly vulnerable.

Compared with adults, children produce proportionately more metabolic heat, have a greater surface area-to-body mass ratio (which causes a greater heat gain from the environment on a hot day), and have a lower sweating capacity, which reduces their ability to dissipate heat through evaporation. These facts emphasize the importance of monitoring heat exposure in children. A fatal event can occur within 20 minutes if normal heat loss mechanisms become overwhelmed. Every year, children left unattended in parked motor vehicles die from heat stroke.

Both children and young adults (most often athletes and laborers) are vulnerable to exertional heat illness, where there has been intense strenuous activity in a hot, humid environment. Elderly, chronically ill, or sedentary adults, as well as children, are vulnerable to nonexertional heat illness. Environmental

Discussion continued

conditions, along with a predisposition for impaired thermoregulation, lead to heat illness in these patients. The elderly and infirm may have diminished cardiac output, a decreased ability to sweat, and decreased ability to vasoregulate. Medications may predispose them to heat illness because of mitigating effects on cardiac output (beta blockers) or on sweating (anticholinergics) or because of volume depletion (diuretics). Nonexertional heat illness may be indolent in its onset and may be associated with significant volume depletion.

Heatstroke is a serious form of heat illness. Treatment, especially aggressive cooling procedures and fluid replacement, must begin immediately to help ensure survival. Morbidity and mortality are directly associated with the duration of elevated core temperature. More intensive evaluation and treatment are required for these patients than is covered in this chapter. The most serious complications of heat stroke are those falling within the category of multiorgan dysfunction syndrome. They include encephalopathy, rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial injury, hepatocellular injury, intestinal ischemia or infarction, pancreatic injury, and hemorrhagic complications, especially disseminated intravascular coagulation, with pronounced thrombocytopenia.

Suggested Readings

American Academy of Pediatrics. (2000). Climatic heat stress and the exercising child and adolescent. *Pediatrics*, *106*(1 Pt 1), 158–159.

Bouchama, A., & Knochel, J. P. (2002). Heat stroke. *New England Journal of Medicine*, *346*, 1978–1988.

Cheshire, W. P. (2016). Thermoregulatory disorders and illness related to heat and cold stress. *Autonomic Neuroscience: Basic and Clinical*, *196*, 91–104.

Wexler, R. K. (2002). Evaluation and treatment of heat-related illnesses. *American Family Physician*, *65*(2307–2314), 2319–2320.

CHAPTER 3

Hyperventilation

Presentation

The patient presenting with hyperventilation syndrome typically appears anxious and exhibits shortness of breath with an inability to fill the lungs adequately. The patient also may have palpitations, dizziness, intense anxiety, fear, chest or abdominal pain, tingling or numbness around the mouth and fingers, and possibly even flexor spasm of the hands and feet (carpopedal spasm) (Fig. 3.1). The patient's respiratory volume is increased, which may be apparent as increased respiratory rate, increased tidal volume, or frequent sighing. The remainder of the physical examination is unremarkable. The patient's history may reveal a precipitating emotional cause or prior similar events. The patient may experience alternating periods of hypoventilation or brief periods of apnea as the body tries to allow carbon dioxide (CO_2) levels to drift back up to the normal range. If this occurs, the pattern is usually abrupt onset of transient apnea without a drop in O_2 saturation, immediately preceded and followed by profound hyperventilation.

What to Do

Perform a brief physical examination, specifically evaluating mental status and listening to breath sounds. Evaluate for evidence of toxins, leg swelling, or other risk factors for pulmonary emboli such as tachycardia or fever.

Measure pulse oximetry, which should be between 98% and 100%, and utilize end-tidal capnometry to guide treatment.

Calm and reassure the patient. Evaluate the patient's ability to follow instructions, and encourage deep, slow breathing. If possible, determine the trigger, if any, to help address the etiology of the event.

If the patient cannot voluntarily reduce ventilatory rate and volume, breathing through a length of tubing (Fig. 3.2) **or a reservoir bag with supplemental oxygen may be utilized.** This will allow the patient to continue moving a large quantity of air but will provide air rich in carbon dioxide (CO_2), allowing the blood partial CO_2 (PCO_2) to rise toward normal. **Administration of 50 to 100 mg of hydroxyzine (Vistaril) intramuscularly (IM) or lorazepam (Ativan) 1 to 2 mg sublingually (SL), IM, or intravenously (IV) often helps to calm the patient, resulting in a reduced respiratory effort.**

If these symptoms cannot be reversed and respiratory effort cannot be reduced in this manner within 15 to 20 minutes, confirm the diagnosis by obtaining arterial blood gas measurements to rule out metabolic acidosis or hypoxia indicative of underlying disease.

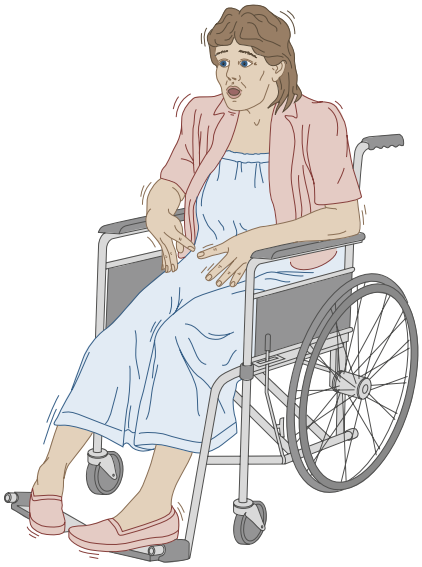

Fig. 3.1 The patient experiences anxiety and shortness of breath and feels as though she is unable to fill her lungs, leading to carpopedal spasm.

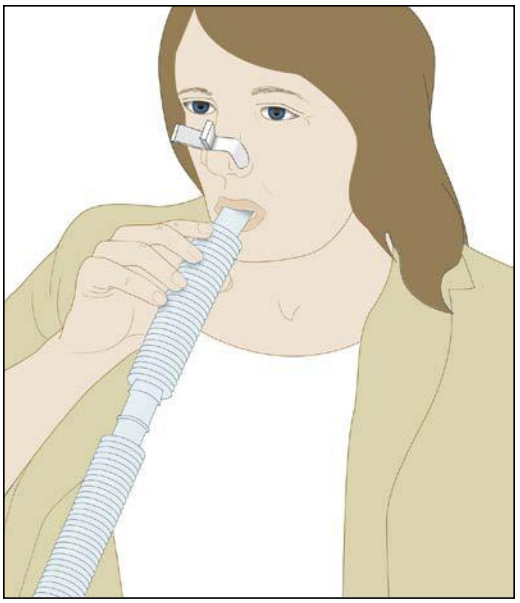

Fig. 3.2 Instruct the patient to breathe through a length of tubing to increase the percentage of inspired CO_2.

Reexamine the patient after hyperventilation is controlled. If possible, identify the psychological stressor that prompted the episode.

Ensure that the patient understands the hyperventilation syndrome and knows some strategies for breaking the cycle next time. Arrange for follow-up with a primary care physician.

What Not to Do

Do not overlook the true medical emergencies, including pneumothorax, asthma, chronic obstructive pulmonary disease (COPD), pneumonia, pulmonary embolus, hyperthyroidism, diabetic ketoacidosis, liver disease, salicylate overdose, sepsis, uremia, substance abuse, sympathomimetic toxidrome, myocardial infarction, congestive heart failure (CHF), and stroke, which also may present with hyperventilation.

Do not use the traditional method of breathing into a paper bag to increase the concentration of inspired CO_2. This increases the potential for inadvertently causing hypoxia and is no longer considered to be appropriate therapy.

Do not do an extensive laboratory and imaging study workup when the history and physical examination are convincingly consistent with psychogenic hyperventilation syndrome. However, be suspicious of an organic cause when the patient has risk factors or does not improve as expected.

Discussion

The acute respiratory alkalosis of hyperventilation causes transient imbalances of calcium, potassium, and other ions, with the net effect of increasing the irritability and spontaneous depolarization of excitable muscles and nerves. Patients with a first-time event of hyperventilation syndrome are the most likely to visit the emergency department, urgent care center, or physician's office, and this is an excellent time to educate them about its pathophysiology and the prevention of recurrence. Patients who have repeated episodes may be experiencing panic attacks, which can be addressed with cognitive therapy or a mild benzodiazepine.

During recovery after hyperventilation, the transition from hypocapnia to normocapnia is associated with hypoventilation. Be aware that patients may experience significant hypoxemia after hyperventilation.

Suggested Readings

Callaham, M. (1989). Hypoxic hazards of traditional paper bag rebreathing in hyperventilating patients. *Annals of Emergency Medicine*, *18*, 622–628.

Chin, K., Ohi, M., Kita, H., et al. (1997). Hypoxic ventilatory response and breathlessness following hypocapnic and isocapnic hyperventilation. *Chest*, *112*, 154–163.

Demeter, S. L., & Cordasco, E. M. (1986). Hyperventilation syndrome and asthma. *American Journal of Medicine*, *81*, 989–994.

Saisch, S. G. N., Wessely, S., & Gardner, W. N. (1996). Patients with acute hyperventilation presenting to an inner-city emergency department. *Chest*, *110*, 952–957.

CHAPTER 4

Psychogenic Nonepileptic Attack

(Dissociative Convulsions)

Presentation

The patient with psychogenic nonepileptic attack (PNEA), also known as psychogenic nonepileptic seizure (PNES), typically presents with the appearance of having tonic-clonic seizure activity or can develop this during a visit.

There may be a history of sexual abuse, eating disorders, depression, substance abuse, anxiety disorders, or personality disorders, and the episode may be preceded by a stressful event. The terminology "pseudoseizure" or "hysterical seizure" is outdated and considered counterproductive as it may give the patient the perception that medical professionals consider this faked or voluntary behavior. Head turning from side to side and pelvic thrusting are common with PNEA, and it may be difficult to determine initially whether the patient is manifesting a true epileptic seizure.

A patient with true seizures usually has abdominal contractions but lacks corneal reflexes, whereas a patient with PNEA usually has corneal reflexes but lacks abdominal contractions. The patient's general color and vital signs are normal, without any evidence of airway obstruction. Consciousness is often partially preserved and sometimes regained very quickly after the convulsive period with PNEA. Commonly, the patient is fluttering the eyelids or resists having the eyes opened. With eyelids closed, a patient with rapid (saccadic) eye movements is awake. On the other hand, a patient with slow, roving eye movements may have a depressed level of consciousness. Tearfulness during the event argues against epileptic seizure (ES). Ictal eye closure is a highly reliable indicator for PNES, while ictal eye opening is generally an indicator of ES.

With PNEA, there is typically no fecal or urinary incontinence, self-induced injury, or lateral tongue biting. Most true seizures are accompanied by a postictal state of disorientation and altered level of arousal and responsiveness. During an epileptic seizure, the plantar response is often extensor, whereas during a PNEA event it is usually flexor.

Noxious stimuli are not reliable in discriminating between PNEA and epileptic seizure. The remainder of the physical examination should be unremarkable.

What to Do

- Obtain any available medical records.
- **Perform a complete physical examination, including a full set of vital signs and O_2 saturation. Patients under the stress of real illness or injury can manifest unusual behavior that may be misinterpreted as seizurelike activity.**

Check glucose with a bedside finger stick.

Monitor vital signs frequently.

When there is significant emotional stress involved, administer a mild tranquilizing agent, such as hydroxyzine pamoate (Vistaril), 50 to 100 mg intramuscularly (IM), or lorazepam (Ativan), 1 to 2 mg intravenously (IV) or IM.

Consider obtaining a drug screen and ask if the patient is safe at home, or has been abused in any way. **In women, consider ordering a pregnancy test.**

If an epileptic seizure is questionable, verify with a lactate level or blood gas analysis, which should show metabolic acidosis with a true epileptic seizure.

When the patient becomes more responsive, reassess, obtain a complete history, and offer follow-up care, including psychological support, if appropriate. PNEA is commonly associated with sexual abuse, eating disorders, depression, substance abuse, anxiety disorders, and personality disorders.

If the patient is not awake, alert, and oriented after about 15 minutes, begin a more comprehensive medical workup. Illnesses to consider include Guillain-Barré syndrome, myasthenia gravis, electrolyte disorders, hypoglycemia, hyperglycemia, renal failure, occult neoplasm, dysrhythmias, systemic infection, toxins, and other neurologic disorders.

What Not to Do

Do not accuse the patient of faking a seizure. Even if the activity is not an epileptic seizure, it is neither voluntary nor is it within the patient's ability to physically control.

Do not attempt to discriminate between PNEA and epileptic seizure with painful stimuli, or by dropping a patient's hand onto the face. These are not discriminatory and may reinforce the patient's history of personal abuse and mistrust of medical professionals.

Do not administer anticonvulsants when PNEA is suspected.

Do not routinely perform extensive workups or become overly aggressive by intubating these patients.

Do not release the patient who has not fully recovered. Instead, the patient must be fully evaluated for an underlying medical problem, which may require hospital admission.

Discussion

PNEA is more common in women than men. In most cases, this represents an involuntary manifestation of psychosocial distress. Antagonizing the patient often prolongs the condition, whereas ignoring the patient seems to take the spotlight off the peculiar behavior, allowing the patient to recover. Some psychomotor or complex partial seizures are difficult to diagnose because of dazed confusion or fuguelike activity and might be labeled as PNEA. The patient might require an electroencephalogram (EEG), administered during sleep, and deserves a referral to a neurologist.

Epilepsy is a common disorder. Psychogenic nonepileptic attack (PNEA) is one of the epilepsy mimics. Video EEG is now the gold standard tool that differentiates between epileptic seizures (ES) and PNEA. Oxygen saturation (SaO_2) and ictal vital signs, including heart rate (HR), respiration rate (RR), body temperature, systolic blood pressure (SBP), and diastolic blood pressure, show crucial changes during ES and PNEA.

The key to successful treatment is earning the trust of the patient to help encourage evaluation and treatment by a behavioral health specialist. This starts with recognizing the patient's symptoms as involuntary and respecting the patient as having true disease that requires a coordinated effort by the larger medical community.

Suggested Readings

Badry, R. (2020). Changes in vital signs during epileptic and psychogenic nonepileptic attacks: A video-EEG study. *Journal of Clinical Neurophysiology*, *37*(1), 74–78.

Benbadis, S. R. (2004). Photo quiz: The value of tongue laceration in the diagnosis of blackouts. *American Family Physician*, *70*, 1757–1758. http://www.aafp.org/afp/20041101/photo.html.

Bounds, J. A. (2007). Ictal eye closure is a reliable indicator for psychogenic nonepileptic seizures. *Neurology*, *68*(12). https://doi.org/10.1212/01.wnl.0000259662.87864.f9.

Dula, D. J., & DeNaples, L. (1995). Emergency department presentation of patients with conversion disorder. *Academic Emergency Medicine*, *2*, 120–123.

Glick, T. H., Workman, T. P., & Gaufberg, S. V. (2000). Suspected conversion disorder: Foreseeable risks and avoidable errors. *Academic Emergency Medicine*, *7*, 1272–1277.

Kaufman, K. R. (2004). Pseudoseizures and hysterical stridor. *Epilepsy and Behavior*, *5*, 269–272.

Reuber, M., Baker, G. A., Smith, D. F., et al. (2004). Failure to recognize psychogenic nonepileptic seizures may cause death. *Neurology*, *62*, 834–835.

Tolchin, B., Martino, S., & Hirsch, L. (2019). Treatment of patients with psychogenic nonepileptic attacks. *JAMA*, *321*(20), 1967–1968.

Viarasilpa, T., Panyavachiraporn, N., Osman, G., et al. (2019). Intubation for psychogenic non-epileptic attacks: Frequency, risk factors, and impact on outcome. *Seizure*, *76*, 17–21.

CHAPTER 5

Idiopathic Facial Paralysis (Bell Palsy)

Presentation

The patient with Bell palsy can present with any of the following signs or symptoms: sudden onset of facial numbness; a feeling of fullness or swelling; periauricular pain; muscular facial asymmetry; an irritated, dry, or tearing eye; drooling; or changes in hearing or taste. Symptoms can develop over several hours or days. Often there will have been a viral illness 1 to 3 weeks earlier, or there may have been another trigger such as stress, fever, dental extraction, or cold exposure. Initial presentation typically includes an isolated partial or complete unilateral facial paralysis in an otherwise alert patient (Fig. 5.1). It is notable that if the forehead paralysis is bilateral, the diagnosis of central cause (stroke) must be considered.

What to Do

Perform a thorough neurologic examination of the cranial and upper cervical nerves and limb strength, noting which nerves are involved and whether unilaterally or bilaterally. **Ask the patient to wrinkle the forehead, close the eyes forcefully, smile, puff the cheeks, and whistle, observing closely for facial asymmetry. Central or cerebral lesions result in relative sparing of the forehead** because of cross-innervation of the orbicularis oculi and frontalis muscles. Check for tearing, eyelid closure, hearing, and, when practical, taste. Observe for corneal desiccation. Examine the ear canal and pinna for herpetic vesicles and the tympanic membrane for signs of otitis media or cholesteatoma.

Patients with facial paralysis accompanied by acute otitis media, chronic suppurative middle-ear disease, mastoiditis, otorrhea, or otitis externa require emergent otolaryngologic consultation.

Facial weakness progressing to paralysis over weeks to months, progressive twitching, or facial spasm suggests a neoplasm affecting the facial nerve.

When facial paralysis is associated with pulsatile tinnitus and hearing loss, suspect a glomus tumor or cerebellar pontine angle tumor.

Diplopia, dysphagia, hoarseness, facial pain, or hypesthesia suggests involvement of cranial nerves other than the seventh and calls for neurologic consultation with early magnetic resonance imaging (MRI).

If there is a history of head trauma, obtain a computed tomography (CT) scan of the head (including the skull base) or an MRI to rule out a temporal bone fracture.

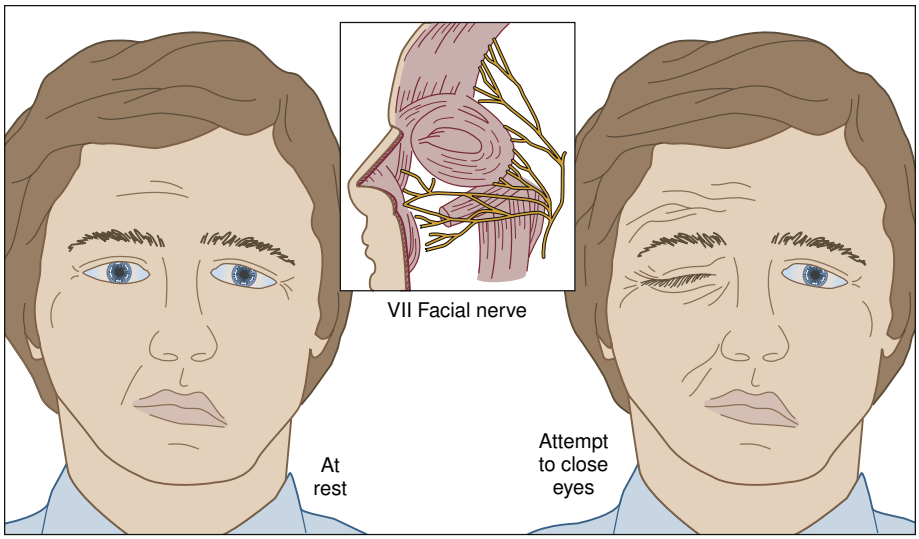

Fig. 5.1. Partial or complete unilateral facial paralysis that includes one side of the forehead in Bell palsy.

MRI with contrast of the skull can reveal lesions, even of small dimensions, inside the temporal bone and at the cerebellopontine angle.

When the findings are consistent with Bell palsy and when there are no absolute contraindications to steroid use, begin therapy with prednisone, 60 mg once daily for 5 days and tapering by 10 mg per day afterward. Prednisone is the only treatment shown to reduce the risk for long-term sequelae of Bell palsy.

Although there is only controversial data to support its efficacy—but because the most widely accepted cause of a true Bell palsy, at present, is a neuropathy induced by herpes simplex virus—**when a patient presents within 7 to 10 days of the onset of acute paresis (or paralysis) and symptoms are severe and no other cause is suspected, it is reasonable to prescribe a 10-day course of either acyclovir (Zovirax), 400 mg five times per day, or 5 days of the more expensive valacyclovir (Valtrex), 500 mg twice daily.** There is some evidence to suggest that treatment within 3 days of the onset of symptoms with combined acyclovir and prednisone therapy may be most beneficial. Again, this is most likely to have the most gain in patients with a complete lesion because they have a higher risk for prolonged facial weakness or other sequelae.

If the cornea is dry or likely to become dry or injured as a result of the patient's inability to produce tears and blink, protect it by patching. If patching is not necessary, recommend that the patient wear eyeglasses, apply methylcellulose artificial tears regularly during the day, and use a protective bland ointment or tape the eyelid shut at night.

When there is a history of a tick bite or rash that is consistent with erythema migrans in areas where Lyme disease is endemic, a doxycycline, 100 mg once daily for 14 days, is indicated. Amoxicillin, 500 mg three times a day for 14 to 21 days, is usually substituted for pregnant

women. Cefuroxime, 500 mg twice daily for 14 to 21 days, and azithromycin, 500 mg once daily for 5 days, have also been used successfully but are generally less effective.

If the cause appears to be herpes zoster varicella or shingles of the facial nerve (e.g., grouped vesicles on the tongue), acyclovir or valacyclovir should still be effective (see Chapter 172). If the geniculate ganglion is involved (i.e., Ramsay Hunt syndrome, with vesicles in or around the ear, decreased hearing, severe otalgia, encephalitis, meningitis), the patient may require hospitalization for intravenous (IV) treatment. The prognosis of Ramsay Hunt syndrome is much worse than that of Bell palsy, with only 10% recovering normal function.

Inform the patient with uncomplicated Bell palsy that symptoms may progress for 7 to 10 days. Of patients with Bell palsy, 70% to 80% recover completely within a few weeks, but patients should be aware of the possibility of permanent facial weakness. Be aware that prognosis is linked to the severity of symptoms. Although most (94%) patients with a partial paralysis recover fully, approximately 40% with a complete paralysis at the time of presentation have some residual weakness. Provide for definite follow-up and reevaluation.

What Not to Do

Do not overlook alternative causes of facial palsy that require different treatment, such as cerebrovascular accidents and cerebellopontine angle tumors (which usually produce weakness in limbs or defects of adjacent cranial nerves), multiple sclerosis (which usually is not painful, spares taste, and often produces intranuclear ophthalmoplegia), and polio (which presents as fever, headache, neck stiffness, and palsies).

Do not order a CT scan unless there is a history of trauma or the symptoms are atypical and include such findings as vertigo, central neurologic signs, or severe headache.

Do not immediately assume that all unilateral facial paralysis is idiopathic facial paralysis. Bell palsy is not likely in patients who report gradual onset of facial paralysis over several weeks or facial paralysis that has persisted for 3 months or more. These patients require further evaluation by a neurologist or an otolaryngologist.

Discussion

Idiopathic nerve paralysis is a common malady, affecting 1 in 60 persons over the course of a lifetime, especially diabetic or pregnant patients and those between the ages of 15 and 45 years. Up to 10% of patients have a recurrence on the ipsilateral or contralateral side. The facial nerve is responsible for facial muscle innervation; lacrimal, nasal, and submandibular gland innervation; taste for the anterior two-thirds of the tongue; and sensation of the external auditory canal, pinna, and tympanic membrane. Although Bell palsy was described classically as a pure facial nerve lesion, and physicians have tried to identify the exact level at which the nerve is compressed, the most common presenting complaints are related to trigeminal nerve involvement. The mechanism is probably a spotty demyelination of several nerves at several sites caused by reactivated herpes simplex virus. Genetic, metabolic, autoimmune, vascular, and nerve entrapment etiologies have been proposed without definitive proof. **Corticosteroids have been shown to improve recovery, and antivirals have demonstrated improved recovery only in severe cases. Eye protection is important for every patient.**

Suggested Readings

Adour, K. K., Ruboyianes, J. M., Von Doersten, P. G., et al. (1996). Bell's palsy treatment with acyclovir and prednisone compared with prednisone alone: A double-blind, randomized, controlled trial. *Annals of Otology, Rhinology & Laryngology*, *105*, 371–378.

Almeida, J. R., et al. (2014). Management of Bell palsy: Clinical practice guideline. *Canadian Medical Association Journal*, *186*(12), 917–922.

Austin, J. R., Peskind, S. P., Austin, S. G., et al. (1993). Idiopathic facial nerve paralysis: A randomized double-blind controlled study of placebo versus prednisone. *The Laryngoscope*, *103*, 1326–1333.

Baringer, J. R. (1996). Herpes simplex virus and Bell's palsy (editorial). *Annals of Internal Medicine*, *124*, 63–65.

Baumgarten, K. L., Lopez, A. A., & Pankey, G. A. (1999). Rash, Bell's palsy, and back pain following a flu-like illness. *Infectious Medicine*, *16*(370–372), 378–379.

Becelli, R. (2003). Diagnosis of Bell palsy with gadolinium magnetic resonance imaging. *Journal of Craniofacial Surgery*, *14*, 51–54.

Grogan, P., & Gronseth, G. (2003). Practice parameter: Steroids, acyclovir, and surgery for Bell's palsy (an evidence-based review). Report of the quality standards subcommittee of the American Academy of Neurology. *Neurology*, *56*, 830–836.

Hato, N., Matsumoto, S., Kisaki, H., et al. (2003). Efficacy of early treatment of Bell's palsy with oral acyclovir and prednisolone. *Otology & Neurotology*, *24*, 948–951.

Murakmi, S., Mizobuchi, M., Nakashiro, Y., et al. (1996). Bell's palsy and herpes simplex virus: Identification of viral DNA in endoneural fluid and muscle. *Annals of Internal Medicine*, *124*, 27–30.

Ronthal, M. (2011). *Bell's palsy: Prognosis and treatment*. UptoDate. http://www.uptodate.com.

Smith, G. N., et al. (2020). Committee Opinion No. 399: Management of tick bites and Lyme disease during pregnancy. *Journal of Obstetrics and Gynaecology (Canada)*, *42*(5), 644–653.

Stankiewicz, J. A. (1987). A review of the published data on steroids and idiopathic facial paralysis. *Otolaryngology-Head and Neck Surgery*, *97*, 481–486.

CHAPTER 6

Migraine Headache

Presentation

Migraine headache is one of the most disabling neurologic disorders, with a lifetime prevalence of 33% in women and 13% in men worldwide. In a third of cases, patients will have an aura, which can manifest with visual phenomena or paresthesias, typically in the hand or oral area. The headache is typically moderate to severe and can be associated with other symptoms such as photophobia, phonophobia, neck pain, nausea, emesis, and cutaneous allodynia. The headache is typically unilateral, throbbing, and worsened by movement, and it commonly lasts 1 to 24 hours or more.

Basilar-type migraine may be associated with fully reversible dysarthria, vertigo, tinnitus, decreased hearing, double vision, or ataxia. Unlike other headaches, migraines are especially likely to wake the patient in the morning. There may be a family or personal history of similar headaches, and onset during the patient's teens or 20s is common. Primary headaches, which include migraine, tension-type headache, and cluster headache, are benign; these headaches are usually recurrent and not caused by organic disease. Secondary headaches are caused by underlying organic diseases, ranging from sinusitis to subarachnoid hemorrhage.

What to Do

Treatment of migraine headache depends on timing. Patients can take preventative treatment, such as beta blockers, antidepressants, calcium channel blockers, angiotensin-converting enzyme inhibitors, or antiseizure medication such as valproic acid. These are of variable effectiveness, and patients can also use alternative methods such as cognitive behavioral therapy or relaxation training to mitigate the occurrence of migraines.

At the onset of headache, patients can take a variety of simple analgesics, such as ibuprofen or acetaminophen, but should be warned against using these medications too often, thereby creating a medication-overuse headache.

If the migraine is moderate to severe in nature, triptans such as sumatriptan (Imitrex), subcutaneously, 1 to 6 mg (may repeat in 1 hour), or intranasally, 1 to 2 sprays (may repeat after 2 hours); intranasal zolmitriptan (Zomig Nasal Spray), 2.5 mg (may repeat every 2 hours up to 10 mg); or any of the oral triptan agents have been shown to be very effective and are considered first-line agents.

If the patient has already used a triptan and presents with a persistent headache, there are many options, including diphenhydramine (Benadryl), 25 to 50 mg IV, ketorolac (Toradol), 30 mg IV, magnesium (an IV infusion of magnesium sulfate), 1 to 2 g in a 10%

solution over 5 to 10 minutes, and/or any of the antiemetics, barring any contraindications individual to the patient. With cost in mind, migraine headaches (and similar recurrent primary headache syndromes, with or without nausea and vomiting) are usually treated successfully with IV prochlorperazine (Compazine), 10 mg (0.15 mg/kg up to 10 mg for pediatric migraine headaches), or metoclopramide (Reglan), 10 mg, with or without a bolus of saline to counteract vasodilatation and orthostasis. **To help prevent mental and motor restlessness (akathisia), administer diphenhydramine (Benadryl), 12 to 25 mg IV, along with the prochlorperazine or metoclopramide.**

If the patient has not taken a triptan, dihydroergotamine (D.H.E. 45) may be used.

Opioids specifically are contraindicated in migraine as they have been shown to perpetuate medication-overuse headaches and opioid-induced hyperalgesia, even with brief administration.

Intranasal 4% lidocaine (Xylocaine) is also an option for resistant migraine. Use a 1-mL syringe. Have the patient lie supine with the head hyperextended 45 degrees and rotated 30 degrees toward the side of the headache, and drip 0.5 mL (10 drops) of the lidocaine solution into the ipsilateral nostril over 30 seconds. The patient should remain in this position for 30 minutes. If the headache is bilateral, repeat on the other side. Another technique is to take 4% lidocaine jelly, apply it to a long cotton pledget, and slide it down the nasal canal using bayonet forceps, posterior to the middle turbinate on the side of the headache. The clinician should be aware that the evidence for the effectiveness of intranasal lidocaine in the acute treatment of migraine is inconsistent.

Clinicians must consider medication efficacy, potential side effects, and potential medication-related adverse events when prescribing acute medications for migraine.

If there are persistent changes in mental status, fever, or stiff neck, or on neurologic examination focal findings such as diplopia or unilateral hyperreflexia, paresthesias, weakness, or ataxia, consider CT, lumbar puncture (LP), or both to rule out intracranial pathology or infection as the cause of the so-called migraine.

Other danger signals that should trigger a more intensive diagnostic workup, looking for secondary disorders, include hyperacute onset of a new severe headache ("the worst ever"); a progressive history of seizures; onset with exertion, cough, bending, or sexual intercourse; onset during pregnancy (cerebral venous thrombosis); and the presence of a systemic malignant disease, infection, compromised immune system, any new neurologic findings, or papilledema on funduscopic examination.

In patients who are older than age 50 years, consider the possibility of temporal arteritis and obtain an erythrocyte sedimentation rate (ESR). If temporal arteritis is present, there may be jaw claudication and tenderness over the temporal artery.

Instruct the patient to return to the emergency department or clinic if there is any change in or worsening of the usual migraine pattern and make arrangements for medical follow-up.

First-time migraine attacks warrant a thorough elective neurologic evaluation to establish the diagnosis.

Long-term prophylaxis may include nonprescription plain magnesium gluconate (200–400 mg three times a day), antidepressants, calcium channel antagonists, nonsteroidal anti-

inflammatory drugs (NSAIDs), beta blockers, or anticonvulsants. Lifestyle changes, such as eliminating caffeine, smoking, and certain food triggers, may also be indicated.

What Not to Do

Do not begin a comprehensive laboratory workup with neuroimaging when the patient presents with a typical benign primary headache with no neurologic deficits.

Do not administer medications containing ergotamine, caffeine, or barbiturates. They are not recommended for continual prophylaxis. They are not effective when used in this manner, and withdrawal from these drugs may actually produce headaches.

Do not fail to provide a definite follow-up appointment, especially for first attacks.

Do not fail to consider the possibility of meningitis, subarachnoid hemorrhage, glaucoma, or stroke; conditions that may deteriorate rapidly if undiagnosed. Patients with subarachnoid hemorrhage who have normal mental status on presentation are at highest risk for misdiagnosis. Do not talk yourself out of doing a CT/LP in any patient with sudden onset (hyperacute) of the worst headache ever just because the patient looks good or has a normal examination.

Discussion

Unilateral pain is even more characteristic of migraine than is the aura. (Migraine is a corruption of the hemicrania continua.) The pathophysiology is probably unilateral cerebral vasospasm (producing the neurologic symptoms of the aura), followed by vasodilation (producing the headache). Neurologic symptoms may persist into the headache phase, but the longer they persist, the less likely it is that they are caused by the migraine. Cluster headaches and other trigeminal-autonomic cephalalgias are characterized by trigeminal activation coupled with parasympathetic activation. These headaches are intermittent, short lasting, sharp, excruciating, and unilateral, accompanied by lacrimation and rhinorrhea. Attacks occur in clusters lasting from 7 days to 1 year, and during the pain, patients are usually agitated and restless. The treatment of an attack is usually the same as that for migraines.

Acute migraine headaches are self-limited and respond well to placebos, and therefore several different therapies are effective. No single drug or class of drug has clearly emerged as the best treatment for acute migraine. The wide variability in patient needs and responses means that many agents will continue to play important roles. Although butalbital-containing compounds are often used to treat migraine, their use should be limited because of the risk of overuse and consequent medication overuse headache and withdrawal problems.

Be cautious in the use of ergot or serotonin agonists to treat any patient who has angina, focal weakness, or sensory deficits. It is possible to precipitate ischemia of the brain or heart in such patients by using preparations that act by causing vasoconstriction. Sumatriptan should not be administered to postmenopausal women, men older than 40 years, and patients with vascular risk factors such as hypertension, hypercholesterolemia, obesity, diabetes, smoking, or a strong family history of vascular disease. Sumatriptan also should not be used within 24 hours of administration of an ergotamine-containing medication.

Fortunately, new options for the acute treatment of migraine attacks are currently in development. As new acute medications become available, it will be important for health care professionals to be aware of current patterns and limitations to medication use and to tailor their approach for migraine treatment to the individual patient, including identifying the appropriate use/combination of acute and preventive pharmacologic and nonpharmacologic treatments.

Discussion continued

Patients with aneurysms or arteriovenous malformations can present clinically as migraine patients. If there is something different about the severity or nature of this headache, consider the possibility of a subarachnoid hemorrhage. Headaches that are always on the same side and in the same location are very suspicious for an underlying structural lesion (e.g., aneurysm, arteriovenous malformation).

To help reassure patients, it can be noted that isolated headache was the first and only clinical symptom in just 8.2% of patients with an intracranial tumor.

Opioids are commonly requested for headache but dangerous in migraine as they can induce hyperalgesia, prevent reversal of migraine central sensitization, and decrease the effectiveness of triptans, which are otherwise a mainstay.

Suggested Readings

Aukerman, G., Knutson, D., & Miser, W. F. (2002). Management of the acute migraine headache. *American Family Physician*, *66*, 2123–2130 2140–2141.

Becker, W. J. (2015). Acute migraine treatment in adults. *Headache*, *55*(6), 778–793.

Brousseau, D. C., Duffy, S. J., Anderson, A. C., et al. (2004). Treatment of pediatric migraine headaches: A randomized, double-blind trial of prochlorperazine versus ketorolac. *Annals of Emergency Medicine*, *43*, 256–262.

Cameron, J. D., Lane, P. L., & Speechley, M. (1995). Intravenous chlorpromazine vs intravenous metoclopramide in acute migraine headache. *Academic Emergency Medicine*, *2*, 597–602.

Charles, A. (2017). The pathophysiology of migraine: Implications for clinical management. *The Lancet Neurology*, *17*, 1–9.

Clinch, C. R. (2001). Evaluation of acute headaches in adults. *American Family Physician*, *63*, 685–692.

Coppola, M., Yealy, D. M., & Leibold, R. A. (1995). Randomized, placebo-controlled evaluation of prochlorperazine versus metoclopramide for emergency department treatment of migraine headache. *Annals of Emergency Medicine*, *26*, 541–546.

Corbo, J., Esses, D., Bijur, P. E., et al. (2001). Randomized clinical trial of intravenous magnesium sulfate as an adjunctive medication for emergency department treatment of migraine headache. *Annals of Emergency Medicine*, *38*, 621–627.

Demirkaya, S., Dora, B., et al. (2001). Efficacy of intravenous magnesium sulfate in the treatment of acute migraine attacks. *Headache*, *41*, 171–177.

Dodick, D. W. (2018). Migraine. *Lancet*, *391*, 1315–1330.

Drotts, D. L., & Vinson, D. R. (1999). Prochlorperazine induces akathisia in emergency patients. *Annals of Emergency Medicine*, *34*, 469–475.

Ferrari, M. D., et al. (2001). Oral triptans (serotonin 5-HT 1B/1D agonists) in acute migraine treatment: A meta-analysis of 53 trials. *Lancet*, *358*, 1668.

Frank, L. R., Olson, C. M., Shuler, K. B., et al. (2004). Intravenous magnesium for acute benign headache in the emergency department. *Canadian Journal of Emergency Medicine*, *6*, 327–332.

Huff, J. S. (1998). What is a migraine, anyway, and when is it gone? *Academic Emergency Medicine*, *5*, 561–562.

Hutchinson, S., Lipton, R. B., Ailani, J., et al. Characterization of acute prescription migraine medication use. *Mayo Clinic Proceedings*, 95(4), 709–718.

Kabbouche, M. A., Vockell, A. B., LeCates, S. L., et al. (2001). Tolerability and effectiveness of prochlorperazine for intractable migraine in children. *Pediatrics*, *107*, e62.

Kao, L. W., Kirk, M. A., Evers, S. J., et al. (2003). Droperidol, QT prolongation, and sudden death: What is the evidence? *Annals of Emergency Medicine*, *41*, 546–558.

Klapper, J. A., & Stanton, J. (1993). Current emergency treatment of severe migraine headaches. *Headache*, *33*, 560–562.

Lipton, R. B., Bigal, M. E., Steiner, T. J., et al. (2004). Classification of primary headaches. *Neurology*, *63*, 427–435.

Maizels, M., Scott, B., Cohen, W., et al. (1996). Intranasal lidocaine for treatment of migraine. *Journal of the American Medical Association*, *276*, 319–321.

Marmura, M. J., Silberstine, S. D., & Schwedt, T. J. (2015). The acute treatment of migraine in adults: The American Headache Society evidence assessment of migraine pharmacotherapies. *Headache*, *55*(1), 3–20.

Matchar, D. B. (DATE). Acute management of migraine. Paper presented at the 55th Annual Meeting of the American Academy of Neurology, Honolulu, Hawaii.

Mauskop, A., Altura, B. T., Cracco, R. Q., et al. (1996). Intravenous magnesium sulfate rapidly alleviates headaches of various types. *Headache*, *36*, 154–156.

Miner, J. R., Fish, S. J., Smith, S. W., et al. (2001). Droperidol vs prochlorperazine for benign headaches in the emergency department. *Academic Emergency Medicine*, *8*, 873–879.

Salomone, J. A., Thomas, R. W., Althoff, J. R., et al. (1994). An evaluation of the role of the ED in the management of migraine headaches. *American Journal of Emergency Medicine*, *12*, 134–137.

Seim, M. B., March, J. A., & Dunn, K. A. (1998). Intravenous ketorolac vs intravenous prochlorperazine for the treatment of migraine headaches. *Academic Emergency Medicine*, *5*, 573–576.

Silvers, S. M., Simmons, B., Wall, S., et al. (2002). Clinical policy: Critical issues in the evaluation and management of patients presenting to the emergency department with acute headache. *Annals of Emergency Medicine*, *39*, 108–122.

Tabata bai, R. R., & Swadron, S. P. (2016). Headache in the emergency department: Avoiding misdiagnosis of dangerous secondary causes. *Emergency Medicine Clinics of North America*, *34*, 695–716.

Tepper, S. (2012). Opioids should not be used in migraine. *Headache*, *52*(Suppl. 1), 30–34.

Vinson, D. R. (2002). Treatment patterns of isolated benign headache in US emergency departments. *Annals of Emergency Medicine*, *39*, 215–222.

Vinson, D. R., & Drotts, D. L. (2001). Diphenhydramine for the prevention of akathisia induced by prochlorperazine: A randomized, controlled trial. *Annals of Emergency Medicine*, *37*, 125–131.

Weaver, C. S., Jones, J. B., & Chisholm, C. D. (2004). Droperidol vs. prochlorperazine for the treatment of acute headache. *Journal of Emergency Medicine*, *26*, 145–150.

CHAPTER 7

Adult Seizures

Presentation

The patient with seizure may present in full tonic-clonic seizure or may have experienced a seizure witnessed by others. Seizure may be preceded by an aura or have sudden onset without warning. Most patients presenting with seizure have a preexisting seizure disorder, and the most common etiology of seizure is noncompliance with medication. Two of the most telling physical exam findings of a patient who has had a seizure include incontinence and lateral tongue biting, but the lack of these signs does not rule out seizure.

What to Do

Many seizures are self-limiting, but if a patient is actively seizing, prepare to administer a benzodiazepine, usually lorazepam (Ativan), either intravenously (IV) or intramuscularly (IM). Observe the seizure activity for patterns, such as eye deviation and/or focal/unilateral presentation.

First-line agents and dosing include the following:

- **1–4 mg IV lorazepam (Ativan)**
- 5–10 mg IV/IM/PR diazepam (Valium) max 5 mg/min (max dose 30 mg)
- 1–5 mg IV/IM/PR midazolam (Versed)

With a prolonged seizure resistant to initial agents, loading with phenytoin (Dilantin) or fosphenytoin (Cerebyx) is recommended to prevent recurrence of seizures. Give phenytoin, 18 to 20 mg/kg IV over 30 minutes, at less than 50 mg/min. (The patient should be on cardiac monitoring during administration, and a Dilantin level should be sent first if the patient is thought to be taking the drug.) Alternatively, give fosphenytoin, 15 to 20 PE/kg IV or IM at a maximum IV rate of 150 PE (phenytoin sodium equivalents)/min with an initial maintenance dose of 4 to 6 PE/min. (Although much more expensive than phenytoin, fosphenytoin can be given more quickly over 15 minutes, or, if IV access is absent, this drug can be given IM; it does not have the tissue toxicity of extravasated phenytoin if IV access is questionable.)

If the patient is still seizing, third-line agents are indicated, including barbiturates, valproate, levetiracetam, and/or propofol in the setting of endotracheal intubation.

Status epilepticus is defined as a generalized tonic-clonic seizure in an adult that lasts more than 5 minutes or intermittent convulsions, without recovery of baseline level of consciousness between seizures.

In all cases of status epilepticus, check the patient's blood glucose level by performing a quick finger stick test and administering IV glucose if the level is below normal.

If the patient arrives in the postictal phase, examine thoroughly for injuries and signs of systemic disease that can provoke seizures. Elevated temperature can be a sign of meningitis or encephalitis. Nuchal rigidity strongly suggests either central nervous system (CNS) infection or subarachnoid hemorrhage. Record a complete neurologic examination. Repeat the neurologic examination periodically, looking for findings suggestive of focal brain disease.

If the patient is indeed recovering, you may be able to obviate much of the diagnostic workup by waiting until the patient is lucid enough to give a history. **Postictal inability to arouse may last 10 minutes after a generalized tonic-clonic seizure, with confusion typically lasting less than 30 minutes.**

If the patient arrives awake and oriented after a presumed seizure, corroborate the history through witness accounts or the presence of injuries, such as a scalp laceration, a bitten tongue, or the presence of urinary or fecal incontinence.

Consider other etiologies of altered mental status if there is no typical postictal recovery period.

Investigate for alcohol or substance abuse; withdrawal from alcohol, benzodiazepines, or barbiturates can provoke seizures.

If the patient has a history of seizure disorder or is taking anticonvulsant medications, determine current and past frequency of seizures. Look for evidence of and reasons for noncompliance, such as loss of insurance or ability to obtain medications.

If the seizure is clearly related to alcohol withdrawal, give 2 mg of IV lorazepam (Ativan) and ascertain why the patient reduced consumption of alcohol. Support the patient's attempt to abstain or detoxify with resources as possible.

If a patient is demonstrating signs of delirium tremens, such as tremors, tachycardia, and hallucinations, withdrawal should be medically supervised and treated with benzodiazepines. Initial treatment with IV lorazepam has been shown to produce a significant reduction in the risk for recurrent seizures related to alcohol.

Because many alcoholics are malnourished, emergency department physicians will often presumptively treat alcohol withdrawal symptoms with an IV infusion containing glucose, 100 mg of thiamine, 2 g of magnesium, 1 mg of folic acid, and multivitamins, even though there is no convincing evidence that this regimen is of any true benefit in isolated alcohol withdrawal. However, **thiamine has been shown to be beneficial in preventing coma and death as a result of Wernicke encephalopathy in patients presenting with altered mental status.** Administration of thiamine and vitamins is inexpensive and has very few side effects. Given this, it is advisable to treat alcoholic patients presenting with acute delirium for both alcohol withdrawal and thiamine deficiency.

If the seizure is a new event, obtain a serum glucose level (to confirm a rapid bedside test result) as well as serum electrolyte concentrations (sodium, calcium, magnesium), renal function tests, hepatic function tests (if liver impairment is suspected), complete

blood cell count (if infection is suspected), and urine toxicology screen (if drugs of abuse are suspected). In women of childbearing age, test for pregnancy.

With new-onset seizures, a brain computed tomography (CT) scan should be performed to rule out intracranial hemorrhage, ischemic stroke, or tumor. Magnetic resonance imaging (MRI) is the gold standard in evaluating seizure disorders and should be obtained when available.

Lumbar puncture should be performed when fever, persistent altered mental status, or nuchal rigidity indicates a possibility of meningitis or encephalitis. Suspicion of subarachnoid hemorrhage should also prompt lumbar puncture, even when head CT scans are normal. A lumbar puncture should also be performed on immunocompromised patients.

About 50% of all patients with a new onset of seizure require hospitalization. Most of these patients can be identified by abnormalities evident on physical examination, head CT scan, toxicology studies, or the other tests mentioned earlier.

If the patient has an established seizure disorder, blood tests are not routinely needed when the patient has a single breakthrough seizure. Anticonvulsant drug levels should be checked when toxicity or noncompliance is suspected. The dose should be adjusted to keep the level above the breakthrough point. Finding a level below the reported therapeutic range should not prompt a dose increase in a patient who has been seizure free for a prolonged period. Neuroimaging and lumbar puncture are unnecessary unless there are new findings to cause suspicion for tumor, intracranial hemorrhage, or CNS infection.

A neurologist should be consulted before antiepileptic drug treatment is initiated for brief new-onset seizures. Many neurologists think it is in the patient's best interest to withhold long-term anticonvulsant therapy until a second seizure occurs. The neurologist may want to make a detailed evaluation of, and counsel the patient regarding, risk for seizure recurrence, the advantages and disadvantages of anticonvulsant therapy, and the psychosocial effect of another seizure. **Patients with a single, brief, uncomplicated seizure, a normal neurologic examination, no comorbidity, and no known structural brain disease need not be started on any antiepileptic drug prior to outpatient referral.**

High risk for recurrence is present when there is a history of brain insult, when an electroencephalogram (EEG) demonstrates epileptiform abnormalities, and when MRI demonstrates a structural lesion.

Patients with generalized seizures should be advised to avoid dangerous situations. They should not swim without supervision and not work at heights. Driving should also be restricted until an appropriate seizure-free period has elapsed, in consultation with a neurologist.

What Not to Do

Do not forget to check blood glucose at the bedside.

Do not fail to be aggressive with the administration of benzodiazepines in the initial phase of treatment. The most common mistake is underdosing or waiting too long. The longer a seizure persists, the greater the risk of status epilepticus.

Do not fail to consult neurology early if the seizure is not aborted with first-line agents.

Do not stick anything in the mouth of a seizing patient. The ubiquitous padded throat sticks may be nice for a patient to hold and to bite on at the first sign of a seizure, but they do nothing to protect the airway and are ineffective when the jaw is clenched.

Do not fail to assume an alcoholic cause. Ethanol abusers sustain more head trauma and seizure disorders than the population at large.

Do not treat alcohol withdrawal seizures with phenobarbital or phenytoin. Both are ineffective (and unnecessary because the problem is self-limiting) and can themselves produce withdrawal seizures.

Do not fail to consider a psychogenic nonepileptic seizure attack (PNEA) in the differential diagnosis and treat appropriately. Do not attempt to apply a noxious stimulus to rule out epileptic seizure. If this is PNEA, it will stop spontaneously, and appropriate management should be instituted (see Chapter 4).

Do not release a patient who has persistent neurologic abnormalities before a head CT scan or specialty consultation has been obtained.

Do not allow a patient who experienced a seizure to drive home.

Discussion

Seizures are time-limited paroxysmal events that result from abnormal, involuntary, rhythmic neuronal discharges in the brain. Except for rare instances, seizures are not predictable and can occur at inconvenient or dangerous times. Seizures are usually short, lasting less than 5 minutes, but can be preceded by a prodromal phase and followed by a long postictal phase, during which there is a gradual return to baseline.

Epilepsy is a disease characterized by spontaneous recurrence of unprovoked seizures. Provoked seizures result from transient alterations in brain metabolism in an otherwise normal brain. Some factors that can trigger such seizures are hypoglycemia, hyponatremia, hypocalcemia, alcohol and medication withdrawal, meningitis, encephalitis, stroke, and certain toxins.

The new terminology for seizures divides them into two classes: generalized seizures and partial seizures. With generalized seizures, there is a complete loss of consciousness at onset of the seizure. Partial seizures are characterized by retained consciousness because they begin in a limited brain region. Partial seizures can secondarily generalize.

There are seven types of generalized seizures, which start throughout the entire cortex at the same time and therefore cause loss of consciousness. They are the following:

1. Generalized tonic-clonic (grand mal) seizures with a tonic phase of whole-body stiffening, followed by a clonic phase of repetitive contractions
2. Tonic seizures, which consist of only the stiffening phase
3. Clonic seizures, which consist of only the repetitive contractions
4. Myoclonic seizures, characterized by brief, lightninglike muscular jerks
5. Absence (petit mal) seizures, which are manifested as brief (1–10 seconds) episodes of staring and unresponsiveness (These seizures, unlike complex partial seizures, are rarely found in adults, are very brief, do not produce postictal confusion, and occur very frequently [up to 100 per day].)
6. Atypical absence seizures, which are similar to absence seizures but last longer and often include more motor involvement
7. Atonic seizures, characterized by sudden loss of muscle tone and subsequent falling or dropping to the floor unprotected (drop attacks) (These seizures must be differentiated from syncope [see Chapter 11].)

Partial seizures are divided into simple and complex. In simple partial seizures, only one neurologic

Discussion continued

modality is affected during the seizure. The resulting symptoms depend on the area of the brain cortex from which the seizure arises. Motor (focal) seizures may produce clonic hand movements. Sensory, autonomic, and psychiatric symptoms may be expressed as visual phenomena, olfactory sensations (usually unpleasant), déjà vu phenomena, and formed hallucinations or memories. These auras are merely simple partial seizures.

Complex partial seizures (psychomotor or temporal lobe seizures) are associated with alteration, but not loss, of consciousness. The patient is awake and staring blankly but is not responsive to external stimuli. These seizures may be accompanied by automatism (repetitive, purposeless movements, such as lip smacking and chewing, hand wringing, patting, and rubbing) and last 30 to 50 seconds. They are followed by postictal confusion and occur weekly to monthly.

The age of the patient is associated with the probable underlying cause of a first seizure and therefore is a factor in disposition. In patients age 12 to 20 years, the seizure is probably idiopathic, although other causes are certainly possible. In the 40-year-old patient experiencing a first seizure, neoplasm, posttraumatic epilepsy, and withdrawal must be excluded. In the 65-year-old patient experiencing a first seizure, cerebrovascular insufficiency must also be considered. With elderly patients, the possibility of an impending stroke, in addition to the other possible causes, should be kept in mind during treatment and workup.

Also, patients should be discharged for outpatient care only if there is full recovery of neurologic function, should possibly be given a full loading dose of phenytoin, and should make clear arrangements for follow-up or return to the emergency department if another seizure occurs. An EEG can usually be done electively, except in cases of status epilepticus. A toxic screen may be needed to detect the many drug overdoses that can present as seizures, including amphetamines, cocaine, isoniazid, lidocaine, lithium, phencyclidine, phenytoin, and tricyclic antidepressants.

Suggested Readings

D'Onofrio, G., Rathlev, N. K., Ulrich, A. S., et al. (1999). Lorazepam for the prevention of recurrent seizures related to alcohol. *New England Journal of Medicine*, *340*, 915–919.

Eisner, R. F., Turnbull, T. L., Howes, D. S., et al. (1986). Efficacy of a "standard" seizure workup in the emergency department. *Annals of Emergency Medicine*, *15*, 33–39.

Foreman, B., & Hirsch, L. J. (2012). Epilepsy emergencies: Diagnosis and management. *Neurology Clinics*, *30*, 11–41.

Henneman, P. L., DeRoos, F., & Lewis, R. J. (1994). Determining the need for admission in patients with new-onset seizures. *Annals of Emergency Medicine*, *24*, 1108–1114.

Huff, J. S., et al. (2014). Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with seizures. *Annals of Emergency Medicine*, *63*, 437–447.

Jagoda, A., & Gupta, K. (2011). The emergency department evaluation of the adult patient who presents with a first-time seizure. *Emergency Medicine Clinics of North America*, *29*, 41–49.

Shneker, B. F., & Fountain, N. B. (2003). Epilepsy. *Disease a Month*, *49*, 426–478.

Towne, A. R., & DeLorenzo, R. J. (1999). Use of intramuscular midazolam for status epilepticus. *Journal of Emergency Medicine*, *17*, 323–328.

CHAPTER 8

Seizures (Convulsions, Fits), Febrile and Pediatric

Presentation

Frightened parents bring in their young child who has just had a first-ever generalized seizure with jerking tonic-clonic movements and loss of consciousness (LOC), followed by a period of postictal obtundation that gradually resolves within 30 minutes. The patient has completely recovered by the time the child is brought to your attention. The parents describe their child becoming cyanotic with breathing difficulty, unresponsiveness, and jerking eye movements during the seizure. The child may be found to have a fever, and there may be a family history of febrile seizures. A vaccination with diphtheria and tetanus toxoids and whole-cell pertussis vaccine may have been administered earlier in the day or 1 to 2 weeks following a measles, mumps, and rubella vaccination.

What to Do

In the Afebrile Child

Begin by assessing the ABCs (airway, breathing, and circulation). Ensure hemodynamic and airway stability in your primary survey, then perform a full secondary survey.

Obtain a history of possible precipitating factors such as trauma or toxin/drug ingestion. Inquire into recent condition(s) and medical history as well as any family history of seizure disorders.

Have witnesses describe the event in detail, including the type of motor and eye movements, changes in breathing and skin color, and whether there was complete LOC or incontinence. Determine the duration of the seizure and the length of the postictal period.

Perform a physical examination that includes evaluation of pupil size and reactivity, and a targeted neurologic examination, including funduscopy to look for retinal hemorrhage, which would suggest intentional injury. After the patient has experienced full recovery from the postictal state, the physical examination should be entirely normal.

Routine laboratory testing other than a screening glucose is usually not needed in children older than 6 months of age, unless there is a history of illness, vomiting or diarrhea, or suspected ingestion.

Infants younger than 6 months of age are at particularly high risk of hypoglycemia; fingerstick glucose testing should be performed as soon as possible. Serum sodium, calcium, and magnesium levels should also be tested to exclude electrolyte disturbance. Toxicology screening should be considered if there is suspicion of toxin exposure.

A computed tomography (CT) scan should be obtained if there are findings of head trauma, a first-time focal (partial) seizure, continuous seizure activity lasting longer than 5 minutes, focal postictal deficits not rapidly resolving (Todd paralysis), persistently altered level of consciousness, sickle cell disease, bleeding disorders, malignancy, or human immunodeficiency virus (HIV) infection. **For most children, immediate neuroimaging is not indicated.**

Children who have one isolated unprovoked seizure—for whom there is no suspicion of trauma, infection, or intoxication—and who have returned to their baseline state may be discharged with appropriate medical follow-up. Antiepileptic drugs (AEDs) are not prescribed.

Parents should be appropriately reassured and informed that 60% of such children never have a recurrence. Discharge instructions should describe what to do if the seizure recurs.

If continuous seizure activity persists for more than 5 minutes, consider bag-valve-mask ventilation or intubation if there is significant respiratory compromise. Intravenous access should be placed, and a bedside glucose test performed.

If the patient is hypoglycemic, 0.5 to 1 g/kg of glucose should be given as a bolus (2 mL/kg of 25% dextrose in water or, in neonates, 5 mL/kg of 10% dextrose in water).

Treatment of status epilepticus should be approached similarly in pediatric and adult patients. First-line treatment should be in the form of a benzodiazepine. Preferred agents vary, depending on the route of administration:

- **With intravenous (IV) or intraosseous (IO) access:**
  - Give lorazepam (Ativan), 0.1 mg/kg IV over 2 to 5 minutes; may repeat in 5 to 10 minutes up to a 4-mg dose (recommended treatment), or
  - Give diazepam (Valium), 0.2 to 0.5 mg/kg IV every 15 to 30 minutes to a maximum 5-mg dose.
- **Without IV or IO access:**
  - Give lorazepam, 0.1 mg/kg per rectum up to a 4-mg dose, or
  - Give diazepam gel (Diastat), 0.5 mg/kg per rectum up to a 10-mg dose, or
  - Give midazolam (Versed), 0.1 to 0.2 mg/kg IM or IN × 1 up to a 10-mg dose.

If status epilepticus persists following one to two doses of a benzodiazepine, then administer an anti-epileptic drug (AED). Note the doses for the recommended agents are generally the same (20 mg/kg) and have a longer duration of action. These are second-line agents and include the following:

- Phenytoin (Dilantin), 20 mg/kg IV at less than 1 mg/kg/min up to 1000 mg
- Fosphenytoin (Cerebyx), 20 mg/kg PE (phenytoin sodium equivalents) up to 1000 mg at less than 3 mg/kg/min (safety and efficacy not established for pediatric patients)
- Phenobarbital, 10 to 20 mg/kg IV up to 1000 mg at less than 1 to 2 mg/kg/min
- Valproate sodium (Depakote), 20 to 40 mg/kg IV over 10 min; 20 mg/kg can be given as a second dose if the patient is still seizing. Goal blood level is 100 mcg/mL and max dose is 3000 mg.
- Levetiracetam (Keppra), 20 to 60 mg/kg with a maximum dose of 4500 mg over 15 minutes