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How It Works

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(Canalith) Otolith
Repositioning Maneuver



Video 2: Eyelid Eversion
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Applicator



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Examination: Normal
Anterior Chamber



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3RD EDITION

Minor Emergencies

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MINOR EMERGENCIES

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To Holly Lindsey

—*Philip Buttaravoli*

To my wife, Robyn, and my children, Zack and Emily, thank you for your love and support. To my colleagues in the Fletcher Allen Emergency Department, thank you for your efforts and hard work on this book and for the outstanding care you deliver to your patients on a daily basis. To Phil Buttaravoli, thank you for the opportunity to participate in the third edition of *Minor Emergencies*.

—*Stephen M. Leffler*

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Foreword

Patients do not experience emergencies that are “minor”; instead, acute care problems and minor urgent problems are “major” to patients, who expect accurate and timely decision making. Moreover, minor emergencies become a challenge to providers if they require an update on management or they need additional information to give the patient the very best care. This textbook details a full repertoire of minor emergencies and is an extremely effective resource in the acute care setting.

Minor Emergencies is a straightforward resource that will aid clinicians on the front line of medicine. The clinical problems are organized by system and are identifiable in the table of contents. Readers will find that it is easy to review a problem, as well as pinpoint and excerpt areas for further consideration. The highlighted discussions succinctly review the pathophysiology or injury mechanism, in addition to the clinical prognosis.

Each section is carefully referenced and contains relevant illustrations, diagrams, and images. Each chapter provides important cautions and highlights steps and strategies for the provider to consider while offering the highest quality care. Each section reflects experience from clinical practice and teaching sessions for learners of acute care medicine. The narrative contains up-to-date scientific approaches that are interwoven with practical strategies. The appendixes contain important protocols and references that will be useful in minor or major emergencies. *Minor Emergencies* is an extraordinary acute care tool for medical students, as well as experienced physicians.

My practice experience ranges from primary care offices in student health settings to athletic fields and the backcountry wilderness and from urgent care settings to the emergency room. On the basis of my experience, I fully recommend this book for the “go-to” shelf of your references. The text is quick, accurate, comprehensive, and effective.

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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 subject 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 and 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 also make them laugh. 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 M. Leffler, MD, 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 of the clinicians out there who are caring for the public's minor emergencies on a daily basis.

Philip 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 Buttaravoli, MD FACEP
(Butter ah'voli)

Stephen M. Leffler, MD FACEP

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I have enjoyed the special opportunity to work with Kate Dimock, who has always been a delight to work with and has been most informative and supportive in the creation of this third edition. In addition, it has been a pleasure working with the extremely competent, efficient, and hardworking assistance of Kate Crowley, Angela Rufino Claire Kramer, and Michael Fioretti, as well as all the other contributors to this project at Elsevier.

Also, special thanks to the emergency department staff at Fletcher Allen Health Care and the University of Vermont.

Neurologic Emergencies

■ Philip Buttaravoli ■ Mario Trabulsy

CHAPTER

1

Dystonic Drug Reaction

Presentation

The patient arrives at the emergency department (ED) or clinic with peculiar posturing, facial grimacing and distortions with a variety of involuntary muscle movements, and/or difficulty speaking. The patient is usually quite upset and worried about having a stroke. 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 are the classic presentations. Often no history is offered. The patient may not be able to speak, may not be aware that he or she took any phenothiazines or butyrophenones (e.g., Haldol that has been used to cut heroin), may not admit that he or she takes illicit drugs 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). Acute dystonias usually present 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
- Tortipelvic—abdominal rigidity and pain
- Opisthotonic—spasm of the entire body

These acute dystonias can resemble partial seizures, the posturing of psychosis, or the spasms of tetanus, strychnine poisoning, or electrolyte imbalances. More chronic neurologic side effects of phenothiazines, including the restlessness of akathisia, tardive dyskinesias, and parkinsonism, do not usually respond as dramatically to drug treatment as do the acute dystonias (Figure 1-1). Onset of oculogyric crisis and torticollis reactions usually occurs within a few minutes or hours but may occur 12 to 24 hours after treatment with a high-potency neuroleptic, such as haloperidol.



Figure 1-1 Patient with dystonic drug reaction.

What To Do:

✔ **Administer 1 to 2 mg of benztropine (Cogentin) or 25 to 50 mg of diphenhydramine (Benadryl) IV**, and watch for improvement of the dystonia over the next 5 minutes. Usually, the medication begins to work within 2 minutes of intravenous 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 on hand in the ED or physician's office. Benztropine may be given to children older than 3 years of age at the dose of 0.01 to 0.02 mg/kg IV, IM, or PO.

✔ **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 2 mg or diphenhydramine 25 mg PO q6h for 24 to 72 hours to prevent a relapse.**

What Not To Do:

- ✘ Do not do any diagnostic workup when findings are typical. Administer benztropine or diphenhydramine first to see if symptoms completely resolve.
- ✘ Do not confuse dystonia with tetanus, seizures, stroke, hysteria, 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, muscle disease).
- ✘ Do not use IV diazepam first because it relaxes spasms resulting from other causes and thus leaves the diagnosis unclear.

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. 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 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 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 SS: Delayed onset of oculogyric crisis and torticollis with intramuscular haloperidol, *Ann Pharmacother* 37:1434–1437, 2003.

Lee AS: Treatment of drug-induced dystonic reactions, *JACEP* 8:453–457, 1979.

Heat Illness

(Heat Edema, Heat Syncope, Heat Cramps, Heat Exhaustion)

2

Presentation

Heat illnesses are 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 edema, 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 edema is dependent edema of the hands and feet that may last for a few weeks.

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, although it too is usually self-limited if treated appropriately. Elderly patients (without air-conditioning on a hot, humid day), workmen, 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 hypotension and painful muscle spasms. The patient may have a normal temperature, 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.**

The severe forms of heat-related illness are **characterized by alteration in mental status** associated with hyperthermia (temperature greater than 40° C). Neuroleptic malignant syndrome and serotonin syndrome are not 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, **which is accompanied by a core temperature of more than 40° C and altered mental status manifested by delirium, seizures, or coma.**

✔ Remove patients with any form of heat illness from the hot environment. Most of the clothing should be removed to promote cooling, and a **rectal temperature should be determined.**

- ✔ Obtain a careful history from the patient or witnesses, with special attention to the type and length of heat exposure, any underlying medical problems, and any medications being used that might predispose the patient to developing heat illness.
- ✔ Perform a physical examination, looking for abnormal vital signs, associated medical illness, dehydration, and diaphoresis.
- ✔ **For heat edema**, inform patients of the benign nature of this problem, and let them know that they can anticipate having this swelling for a few weeks. Advise them to keep their extremities elevated above the level of their heart as much as possible and, in severe cases, to use compressive stockings.
- ✔ **For heat syncope or presyncope**, patients should rest and receive oral or intravenous rehydration. They should be thoroughly evaluated for any injury resulting from a fall, and **all potentially serious causes of syncope should be considered** (see Chapter 11).
- ✔ **For heat cramps** alone, provide muscle stretching and massage, and administer an oral electrolyte solution ($\frac{1}{2}$ tsp table salt in 1 quart of water) or intravenous normal saline for rapid relief.
- ✔ **For true heat exhaustion**, provide intravenous rehydration with normal saline or a glucose-in-hypotonic saline solution (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. Electrolyte abnormalities should be corrected appropriately. Rapid correction of hypernatremia can cause cerebral edema.
- ✔ **When there is hyperthermia**, the patient should be sprayed or sponged with tepid or warm water (to prevent shivering) and then fanned to enhance evaporation and cooling. Ice water immersion is more effective in rapid cooling but poorly tolerated in most patients (especially elderly patients).
- ✔ If not treated properly, **heat exhaustion may evolve into heatstroke, which is a major medical emergency that may lead to cardiac arrhythmias, rhabdomyolysis, serum chemistry abnormalities, disseminated intravascular coagulation, irreversible shock, and death**. Physical examination and laboratory analysis should provide the correct diagnosis.
- ✔ When **patients with minor forms of heat illness** respond successfully to treatment, with vital signs returning to normal and symptoms relieved, they 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 should be encouraged to maintain adequate fluid intake in the future, to prevent recurrence. People engaged in strenuous exercise in hot weather should be encouraged to drink water more frequently than thirst dictates. Runners should drink 100 to 300 mL of water or a hypotonic glucose-electrolyte solution (Gatorade and others) 10 to 15 minutes before beginning a race and should drink about 250 mL every 3 to 4 kilometers. 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 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.
- ✘ Do not allow overhydration in athletes who are trying to prevent heat illness (especially women and slow runners). Severe cases of hyponatremia that resulted from excessive water consumption have been reported.

Discussion

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 danger of leaving a child unattended in a car during hot weather. A fatal event can occur within 20 minutes if normal heat loss mechanisms become overwhelmed.

(continued)

Discussion continued

Both children and young adults (most often athletes and laborers) are associated with exertional heat illness, where there has been intense strenuous activity in a hot, humid environment. Elderly, chronically ill, or sedentary adults, as well as small children, are associated with nonexertional heat illness. Environmental 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 negative 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 the deadliest of heat illnesses. 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: Climatic heat stress and the exercising child and adolescent, *Pediatrics* 106 (1 Pt 1):158–159, 2000.

Bouchama A, Knochel JP: Heat stroke, *N Engl J Med* 346:1978–1988, 2002.

Wexler RK: Evaluation and treatment of heat-related illnesses, *Am Fam Physician* 65:2307–2314, 2319–2320, 2002.

Hyperventilation

Presentation

The patient is anxious and complains of shortness of breath and 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) (Figure 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 normal. The patient's history may reveal an obvious precipitating emotional cause (such as having been caught stealing or being in the midst of a family quarrel or any other form of stress during work or normal life). The patient may experience alternating periods of hypoventilation or brief periods of apnea as her body tries to allow carbon dioxide (CO₂) 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 O₂ saturation, immediately preceded and followed by profound hyperventilation.

What To Do:

- ✔ Perform a brief physical examination, checking especially that the patient's mental status is good; there is no unusual breath odor; there are good, equal excursion and breath sounds in both sides of the chest; and there is no swelling, pain, or inflammation of the legs or other risk factors for pulmonary emboli.
- ✔ **Measure pulse oximetry**, which should be between 98% and 100%.
- ✔ **Calm and reassure the patient.** Explain to the patient the cycle in which rapid, deep breathing can cause physical symptoms upsetting enough to cause further rapid, deep breathing. Repeat a cadence ("in... out... in...") to help the patient voluntarily slow her breathing, or have her voluntarily hold her breath for a while.
- ✔ **If the patient cannot reduce her ventilatory rate and volume, provide a length of tubing through which she can breathe (Figure 3-2), or use a reservoir bag with O₂, keeping the pulse oximetry monitor on to avoid hypoxia.** This will allow the patient to continue moving a large quantity of air but will provide air rich in carbon dioxide (CO₂), allowing the blood partial CO₂ (Pco₂) to rise toward normal. (Carbogen gas [5% CO₂] also may be used, if available.) **Administration of 50 to 100 mg of hydroxyzine (Vistaril) IM or lorazepam (Ativan) 1 to 2 mg SL, IM, or IV often helps to calm the patient, allowing her to control her respirations.**

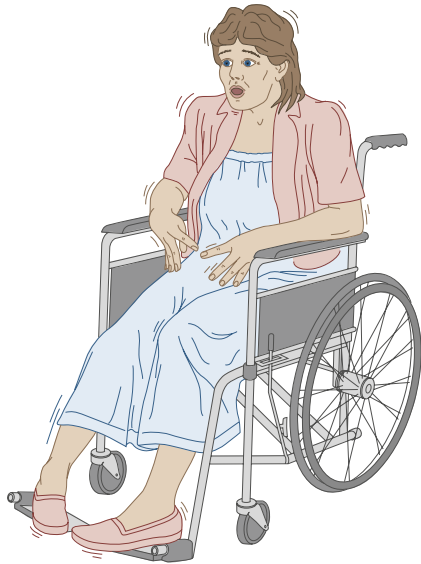


Figure 3-1 The patient experiences anxiety and shortness of breath and feels as though she is unable to fill her lungs, leading to carpopedal spasm.




Figure 3-2 Instruct the patient to breathe through a length of tubing to increase the percentage of inspired CO_2 .

✔ **If these symptoms cannot be reversed and respiratory effort cannot be reduced in this manner within 15 to 20 minutes, double check the diagnosis by obtaining arterial blood gas measurements and looking for a metabolic acidosis or hypoxia indicative of underlying disease.**

- ✔ Reexamine the patient after hyperventilation is controlled. Identify the psychological stressor that prompted the attack.
- ✔ Ensure that the patient understands the hyperventilation syndrome and knows some strategies for breaking the cycle next time. (It may be valuable to have the patient reproduce the symptoms voluntarily). Arrange for follow-up with a primary care physician or psychiatric counselor as needed.

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 perhaps other ions, with the net effect of increasing the irritability and spontaneous depolarization of excitable muscles and nerves. First-time victims of the hyperventilation syndrome are the most likely to visit the emergency department or doctor's office, and this is an excellent time to educate them about its pathophysiology and the prevention of recurrence. Repeat visitors may be overly excitable or may have emotional problems and need counseling.

During recovery after hyperventilation, the transition from hypocapnia to normocapnia is associated with hypoventilation. Be aware that patients may experience significant hypoxemia after hyperventilation. Some investigators believe that there is no benefit in having a patient rebreathe her own exhaled air and that any benefit provided is the result of the reassurance of "instructional manipulation" and the patient's belief in the treatment rather than the elevated fractional concentration of CO₂ in inspired gas (FiCO₂).

Suggested Readings

Callaham M: Hypoxic hazards of traditional paper bag rebreathing in hyperventilating patients, *Ann Emerg Med* 18:622–628, 1989.

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

Demeter SL, Cordasco EM: Hyperventilation syndrome and asthma, *Am J Med* 81:989–994, 1986.

Saisch SGN, Wessely S, Gardner WN: Patients with acute hyperventilation presenting to an inner-city emergency department, *Chest* 110:952–957, 1996.

Hysterical Coma or Pseudoseizure

Presentation

The patient is unresponsive and brought to the emergency department on a stretcher. There is usually a history of recent emotional upset: an unexpected death in the family, school or employment difficulties, or the breakup of a close relationship. There may be a history of sexual abuse, eating disorders, depression, substance abuse, anxiety disorders, or personality disorders. Hysterical coma and pseudoseizures rarely occur in social isolation. The patient may be lying still on the stretcher or demonstrating bizarre posturing or even asynchronous or dramatic thrashing with prolonged seizure-like movements. Head turning, from side to side, and pelvic thrusting are typical of psychogenic seizures. A patient with true seizures usually has abdominal contractions but lacks corneal reflexes, whereas a patient with pseudoseizures 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. Commonly, the patient is fluttering his or her eyelids or resists having his or her 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 has a true depressed level of consciousness. Tearfulness during the event argues against true epileptic seizure. With pseudoseizures, there should not be 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 psychogenic nonepileptic seizure, it is usually flexor.

A striking finding in hysterical coma is that the patient may hold his or her breath when the examiner breaks an ammonia capsule over the patient's mouth and nose. (Real coma victims usually move the head or do nothing.) A classic finding in hysterical coma is that when the patient's apparently flaccid arm is released over his or her face, it does not fall on the face but drops off to the side. The patient may show remarkably little response to painful stimuli, but there should be no true focal neurologic findings, and the remainder of the physical examination should be normal.

What To Do:

- ✓ Obtain any available medical records.
- ✓ **Perform a complete physical examination, including a full set of vital signs and O₂ saturation. Patients under the stress of real illness or injury sometimes react with hysterical or histrionic behavior.** This is especially true in patients with a history of psychiatric illness, substance abuse, or sociopathic behavior. Therefore always fully investigate any suspicion of true underlying pathology.

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