

Examining American Medical Education Priorities After 9/11

To the Editor: The terrorist attacks on September 11, 2001, and the subsequent cases of US mail-disseminated anthrax infection have profoundly changed our society. Widely read large-circulation medical journals such as *Mayo Clinic Proceedings* have published original research, review articles, and guidelines to help practicing clinicians more effectively identify weapons of mass destruction (WMD) attacks and initiate appropriate responses,¹⁻³ but these publications alone are not sufficient. To complement these efforts, the Association of American Medical Colleges—while acknowledging that several medical schools have implemented some training for WMD attacks—has indicated the need for “guidance regarding the content and teaching methods that would be most appropriate” for learners.⁴ At present, a variety of continuing medical education (CME) activities are available to train clinicians regarding WMD, and specialty societies, among others, are developing various additional CME resources, Internet-based links, and algorithms. WMD-related knowledge and skills must be incorporated into the training of physicians at all levels, from medical school and graduate education to CME and board recertification. We need to ask ourselves, “Are we doing enough?”

We believe that WMD-related instruction should follow the model of basic life support or advanced cardiac life support programs, with introduction in the undergraduate curriculum and reinforcement through a career-long recertification program. This recommendation is based on the understanding that dealing with WMD injuries requires unique knowledge and skills that, once acquired, may deteriorate with time if not periodically reviewed and practiced. The need for expertise in the medical treatment of WMD injuries is no longer unique to military physicians, and the experience at the Uniformed Services University School of Medicine may provide a model for others. At the Uniformed Services University, basic science courses incorporate topics such as the pathology of biowarfare agents, the physiology of radiation injuries, and pharmacological treatments for WMD attacks. Furthermore, every student participates in hands-on training to inculcate skills such as triage, decontamination, self-protection in a WMD environment, and medical planning for emergencies. Two week-long field exercises, one in the summer before the second year of medical school and the other during the fourth year, mimic a real-world experience for students in all aspects of WMD medical treatment.

Instruction regarding WMD injuries should not remain confined to undergraduate medical education. Graduate medical education optimally ensures that all physicians have the ability to recognize the clinical presentations of WMD attacks. Unfortunately, a recent study found that only 1 in 4 physicians surveyed feel comfortable in this regard.⁵ Moreover, only 18% of the physicians reported receiving prior WMD training, although 93% expressed a desire for such education. Military hospitals have already established educational objectives and integrated training courses⁶ (eg, a 3- or 6-day Medical Man-

agement of Chemical and Biological Casualties Course) and operational rotations into the academic residency training. Similar courses could also be used to prepare clinicians in civilian training programs to deal with this threat.

When we become physicians, we take an oath to ensure that we have the tools and training necessary to treat the health threats that afflict our patients. In the 21st century, this includes WMD. It is time to reexamine our priorities for medical education.

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A Car Crash Complicating Influenza

To the Editor: Influenza A virus causes an acute respiratory infection characterized by tracheobronchitis and systemic illness. Common symptoms of influenza include fever, myalgias, fatigue, and cough. Most patients recover uneventfully, but some patients may experience complications including pneumonia, rhabdomyolysis, or encephalitis. I report an unusual complication of acute influenza A infection, a motor vehicle crash resulting from cough-induced syncope.

Report of a Case. A healthy 45-year-old man had acute onset of fever, chills, myalgias, and a nonproductive cough. The cough was severe and prolonged at times, with 1 paroxysm producing severe light-headedness followed by a brief loss of consciousness. On the second day of the illness, the patient experienced a severe coughing paroxysm while driving home from work. He felt extremely light-headed and nauseous and abruptly lost consciousness; his car crossed 4

lanes of traffic and hit a hillside. The patient was admitted to the hospital with several non-life-threatening orthopedic injuries. He was febrile and complained of nonproductive cough and myalgias. Physical examination revealed an obese man with scattered wheezes and trauma-induced ecchymoses. A direct fluorescent antibody test performed on a nasopharyngeal swab specimen was positive for influenza A virus; chest radiography revealed no infiltrate. The patient was treated supportively and was discharged from the hospital without incident. He was counseled to avoid driving during subsequent respiratory infections associated with coughing.

Discussion. Cough syncope, first described by Charcot in 1876, classically occurs in moderately obese, middle-aged men, similar to the patient described in this report.¹ In fact, in 1 series, 97% of 270 cases of cough syncope occurred in males,² and these patients are often described as “hearty, robust, and large chested.”¹ Why obesity and male sex predispose to cough syncope is unclear, but redundant oropharyngeal tissue could result in transient airway obstruction and subsequent hypoxia during vigorous coughing. In addition, during a coughing paroxysm, intrathoracic pressure increases and obstructs venous outflow, culminating in an acute decline in cardiac output, blood pressure, and cerebral perfusion that leads to loss of consciousness.¹ Another proposed mechanism of cough syncope is decreased cerebral blood flow as a consequence of elevated cerebrospinal fluid pressure from acute increases in cerebrovascular resistance.³ In addition, reflex-induced bradycardia or heart block may occur in some patients. Rarely, coughing may precipitate syncope in the presence of a hypersensitive carotid sinus.⁴ Because syncope that occurs during driving may lead to devastating consequences, physicians should counsel patients (especially

overweight males) with influenza or other respiratory illnesses to avoid driving or to pull off the road if they experience uncontrollable coughing.

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CORRECTION

Incorrect information: In the article by Shanafelt et al entitled “Pathophysiology and Treatment of Hot Flashes,” published in the November 2002 issue of the *Mayo Clinic Proceedings* (*Mayo Clin Proc*. 2002;77:1207-1218), an incorrect statement was printed in the sixth through ninth lines of the “Neurotransmitters as Effectors of Hot Flashes” section on page 1209, and an incorrect symbol was printed in Figure 1 on page 1209. The sentence should read, “Estrogen withdrawal is associated with decreased blood serotonin levels, **whereas estrogen increases serotonin receptors in the hypothalamus.**” In Figure 1, the symbol between the “estrogen” and “receptor concentration” boxes should be a “+” rather than a “-”.

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