When Most Doctors Are Women

TO THE EDITOR: It goes without saying that Levinson and Lurie’s Perspective, “When Most Doctors Are Women: What Lies Ahead?” (1), has created a great deal of interest and, particularly with physicians who are women, a great deal of discussion. My choice of words in the previous sentence, however, really highlights the problem—these women are physicians. We should not attach the label “women physicians.” We certainly have never used the label “men physicians.”

A major disappointment in Levinson and Lurie’s article was the lack of hard data or even very specific studies. Our major concern for the future should be that we continue to have enough physicians who are very well trained and dedicated to their profession. Physicians are people, with the varied interests, skills, and abilities that exist in the human race. It is most likely true that a smaller proportion of physicians who happen to be women choose the strenuous and ambitious careers available in medicine and science. What we must safeguard is the right of physicians who are women to choose such pathways if they so desire.

Certainly, when a greater proportion of physicians and scientists are men, wonderful things were achieved. More recently, there have been some wonderful achievements by women in medicine and science. Other influences probably now affect both men and women who have made dedicated commitments to their profession and science. Overall, we have a more relaxed approach to living than have previous generations, enjoying the arts, sports, and family. More particularly for physicians, many of the recent requirements of the Accreditation Council for Graduate Medical Education may curtail the contributions of young physicians in their postdegree training, both as residents and fellows. These regulations are affecting career choices and commitments for both men and women.

Last, Levinson and Lurie commented that in Russia and other countries, “medicine has long been dominated by women.” It should be recognized that overall the training of physicians in these countries was not of the highest standards; hospitals and facilities were very poor. Therefore, these comments were not relevant. The authors do admit that various problems have not yet been adequately analyzed.

I and other physicians at my institution who are women hope that the publication of Levinson and Lurie’s Perspective will provide a stimulus for thinking and debate. We also hope it will yield some good advice that can be used by all physicians, both men and women.

Evelyn V. Hess, MD
University of Cincinnati Medical Center
Cincinnati, OH 45267-0563

Reference

Breast Cancer after Childhood Cancer

TO THE EDITOR: I read with great interest the article by Kenney and colleagues (1) on risk factors associated with development of secondary breast cancer in a population of childhood cancer survivors. Were the authors able to perform their analysis taking into account the patients’ birthweights? Recently, Ahlgren and colleagues (2) emphasized the already recognized role of high birthweight as an independent risk factor for breast cancer. Moreover, several studies (3–5) have indicated a positive association between high birthweight and the risk for some types of cancer (for example, Wilms tumors, brain tumors, leukemia, and lymphoma). Theoretically, therefore, in the context of Kenney and colleagues’ population, in which patients developed breast cancer after having childhood cancer, it could be argued that high birthweight could somehow influence the genesis of both tumors in the same patient. It should be of interest to determine whether high birthweight may be considered an independent risk factor for secondary breast cancer. In positive cases, clinicians would have a novel way to identify a specific subgroup of childhood cancer survivors who might benefit from early, vigilant screening for breast cancer.

Gabriele Rossi, MD
University of Pavia
27100 Pavia, Italy

References

TO THE EDITOR: Regarding the findings of Kenney and colleagues (1), it is my hypothesis that low levels of dehydroepiandrosterone (DHEA) may trigger breast cancer, and that this phenomenon is often accompanied and caused by high testosterone levels (2). My hypothesis has received demonstrable support. Hormone replacement therapy (HRT) increases the probability of breast cancer. I suggest this occurs because HRT and estrogen replacement therapy both reduce DHEA levels (3). Also, “androstenedione and testosterone might be more strongly associated with [breast cancer] risk than estradiol” (4). Testosterone reduces DHEA levels. Stahlberg and associates (5) reported, “In current users of combined HRT with testosterone-like progestins, the continuous combined regimens were associated with a statistically significant higher risk of breast cancer than the cyclical combined regimens.” I suggest the reason for the increased risk caused by the “testosterone-like progestins” may be due to the possibility that they act like testosterone and reduce overall DHEA levels.
It is also my hypothesis that the “secular trend,” the increase in size and earlier puberty in our children, is actually an increase in the percentage of individuals with higher testosterone levels in our populations. The secular trend is real and robust in the United States (6). More specifically, it is the exposure of fetuses to higher levels of testosterone in utero that is causing the secular trend, including the increase in breast cancer.

Kenney and colleagues report risk factors for “breast cancer among female survivors of childhood cancer” that may also support my hypothesis. Total-body irradiation of girls significantly reduced DHEA levels up to 5 years after treatment (7). Conversely, in rats, “DHEA has a potent preventive activity against the promotion/progression phase of radiation-induced tumorigenesis” (8). Hypothyroid conditions and hyperthyroid conditions are accompanied by decreases and increases, respectively, in DHEA level. Pelvic radiation therapy frequently results in ovarian failure (9). This may decrease the production of testosterone in this cohort and, therefore, reduce the effects of testosterone in reducing DHEA level. The protective effect of pelvic radiation reported by Kenney and colleagues may be due to reduced testosterone levels.

Kenney and colleagues’ findings may represent various phenomena in their cohort that reduce DHEA levels and, therefore, increase breast cancer. One exception is pelvic radiation, which may reduce the negative effect of testosterone, thereby increasing DHEA levels.

James M. Howard, BS
Fayetteville, AR 72701

References

IN RESPONSE: One of the objectives of our study was to investigate how hormonal and reproductive factors, known to modify breast cancer risk in the general population, modify secondary breast cancer risk in the unique population of childhood cancer survivors. We acknowledge that breast carcinogenesis is complex and that ovarian steroid hormones are not the only factors that contribute to an individual’s breast cancer risk. However, in our attempt to identify risk factors for secondary breast cancer beyond radiation treatment, we chose to focus on hormonal and reproductive factors that are well established as breast cancer risk factors for the general population (1). In addition, our analysis was limited to data that were available on this cohort. Future studies on secondary breast cancer risk in childhood cancer survivors might address the potential risk factors that Dr. Rossi and Mr. Howard mention.

Lisa B. Kenney, MD, MPH
Dana-Farber Cancer Institute
Boston, MA 02115

Evidence for Expanding Physician Supply

TO THE EDITOR: Richard Cooper, in his thoughtful and well-referenced article on physician supply (1), cites cardiology as a specialty confronting a mismatch in the supply of and demand for doctors. Several scientific, technological, social, and demographic factors are driving demand for cardiologists. Meanwhile, the output of cardiology training programs decreased significantly in the 1990s (2). The American College of Cardiology (ACC) recently published a detailed report of a 2-year study of the cardiology workforce (3). Titled “Cardiology’s Workforce Crisis: A Pragmatic Approach,” the ACC report includes sections devoted to 1) the origins and implications of a growing shortage of cardiologists, 2) ways to increase the supply of cardiologists, 3) how to encourage more women and underrepresented minorities to choose a career in cardiology, 4) the growing number of international medical graduates in cardiology, 5) how cardiologist-led teams of nonphysician clinicians can enhance cardiovascular care, 6) the role of technology in enhancing efficiency, 7) methods to improve the job-matching process, and 8) how to encourage more cardiology trainees to choose a career in general clinical cardiology. The report includes several specific recommendations to help address the growing shortage of cardiovascular specialists. It is to be hoped that Cooper’s paper and studies such as the ACC-sponsored workforce report will act as a catalyst for academic medical centers, regulatory organizations, federal policymakers, professional societies, and other organizations that influence the output of cardiologists to address this problem. As the national burden of cardiovascular disease continues to grow, we must increase the output of cardiologists who will devote their careers to prevention, early and accurate diagnosis, and cost-effective treatment of cardiovascular disease.

W. Bruce Fye, MD, MA
Mayo Clinic College of Medicine
Rochester, MN 55905

References
TO THE EDITOR: In contrast to what Cooper’s article (1) suggests, the root of the problem of physician undersupply in the coming years is not only the limits on allopathic medical school admissions following the 1970s but also the drastic changes in the health care world that have made becoming a doctor a headache-ridden nightmare. Today’s doctors must deal with a changing health care climate in which insurance carriers dictate how much they can make and how they practice medicine. Look at the recent report in Medical Economics (2) detailing how physician salaries, especially those of much-needed primary care physicians, trail way behind rising inflation costs and the costs of running a practice in this age of added regulations, the Health Insurance Portability and Accountability Act (HIPAA), and so on.

I know of numerous medical school and residency friends who have left the medical service field of caring for patients because of the increasing unattractiveness of efforts made versus rewards received. This is not only due to limited spaces for medical school admission; this is due to an overworked health care force telling their youth to find another career that is more attractive in terms of hours and rewards. Who wants to be on the phone all day getting precertification for this and that, or being told when and how a test may be ordered? I’m sorry. The solution is far more complex than merely creating more “spaces” in allopathic medical schools across the United States. We need to fix the root of the problem: an insurance system devoid of the necessary checks and balances from the proper authorities. We need to give medicine back to the physicians and patients, trail way behind rising inflation costs and the costs of running a practice in this age of added regulations, the Health Insurance Portability and Accountability Act (HIPAA), and so on.

I know of numerous medical school and residency friends who have left the medical service field of caring for patients because of the increasing unattractiveness of efforts made versus rewards received. This is not only due to limited spaces for medical school admission; this is due to an overworked health care force telling their youth to find another career that is more attractive in terms of hours and rewards. Who wants to be on the phone all day getting precertification for this and that, or being told when and how a test may be ordered? I’m sorry. The solution is far more complex than merely creating more “spaces” in allopathic medical schools across the United States. We need to fix the root of the problem: an insurance system devoid of the necessary checks and balances from the proper authorities. We need to give medicine back to the physicians and patients, trail way behind rising inflation costs and the costs of running a practice in this age of added regulations, the Health Insurance Portability and Accountability Act (HIPAA), and so on.

Vincent M. Pedre III, MD
Mount Sinai Medical Center
New York, NY 10028

References
The need for critical care services will grow as the baby boomers retire, but the number of trained critical care physicians is not growing. A joint project of the American Thoracic Society, the American College of Chest Physicians, and the Society of Critical Care Medicine assessed the supply and demand and projects a major (almost 50%) shortfall in providers by 2010 (3). This predicted shortfall was only slightly delayed but was not prevented by the unlikely scenario of a reduction of critical care services to the elderly.

The critical care community is feeling the physician shortage today. We expect the rest of the physician community will feel the shortage in the near future, and it will dictate changes in the health care system. Whether the answer is to increase the supply of physicians or mid-level practitioners, use more technology, adjust the price of health care, or a blend of all of these options, it is important that we make policy choices today so we can avoid a system breakdown in the very near future.

Sharon I.S. Rounds, MD
Derek C. Angus, MD, MPH
Brown Medical School
Providence, RI 02908

References

IN RESPONSE: Dr. Fye and his colleagues at the ACC are to be congratulated for calling attention to the plight of cardiology or, more correctly, the plight of patients who will need cardiologists but who, because of shortages, will lack the opportunity to see them. It’s a sad day when Americans cannot access the advanced cardiac care that our nation has invested so heavily in creating while billions are squandered on regulation, litigation, and other administrative intrusions. These not only add cost but, as Dr. Pedre notes, also create bureaucratic burdens that force physicians to leave practice altogether. It is also sad that, while physician shortages were evolving, the Council on Graduate Medical Education was steadfast in forecasting impending physician surpluses, not because, as Drs. Curry and Barganier suggest, the AMA Masterfile is wrong (although it is far from perfect), but rather because the Council’s methodology was wrong. Indeed, the Council used the same AMA data in its more recent model, fashioned after our own, which caused it to reverse course and acknowledge that the problem was one of shortages rather than surpluses (1).

In their accompanying editorial, Drs. Garber and Sox (2) sought comfort in the notion that the United States may not need more physicians because older folks are healthier these days and don’t require as much care. Of course, they’re healthier because of their stents, artificial hips, and cataract operations, and despite these aids, they’re certain to encounter disease and its costly therapy still later in life. But most of this care isn’t even necessary, at least if one accepts the “supplier-induced demand” argument, as Drs. Garber and Sox have done. This notion springs from an observed correlation between the number of surgeons and the amount of surgery, but a similar correlation exists between obstetricians and babies (3), so where does the demand originate? And lest anyone think that the advances of the past 3 decades have added value, the editorialists dutifully recite the “more is worse” catechism (4), a statistical artifact of population clustering. Most terrifying is their notion that price will control demand, a disastrous scenario in which shortages of physicians will lead to such steep increases in cost that use will fall because most people won’t be able to afford care, so why produce more physicians anyway? One can only hope that reason will prevail.

Richard A. Cooper, MD
Medical College of Wisconsin
Milwaukee, WI 53226

References

Drug-Induced Thrombocytopenia: An Updated Systematic Review

TO THE EDITOR: We previously systematically reviewed all English-language reports, published up to 1 August 2002, on drug-induced thrombocytopenia (1–3). Our goal for continuing to update our database is to provide an accessible resource (http://moon.ouhsc.edu/george) for all reports of drug-induced thrombocytopenia, describing the level of evidence for a causal role for each drug as well as clinical outcomes (1). This letter describes our continuing systematic review for the period from 2 August 2002 to 14 August 2004.

We reviewed both individual-patient data and group data presented in case series and clinical trials to assess the probability of drug-induced thrombocytopenia. Through a MEDLINE search, using our previously described strategy (1), we retrieved 70 articles; we identified 58 additional articles by searching the bibliographies of the retrieved articles. Fifty-one of the 58 additional articles had been published between 1966 and 1 August 2002 but had not been identified in our previous searches (1–3), again demonstrating the incompleteness of literature searches. Using our previously published criteria (1), 2 of the authors independently reviewed each patient case report to establish the level of evidence for a causal role of the drug in thrombocytopenia. Seventy-four articles contained 103 case reports of individual-patient data, of which 39 were excluded because they did not meet previously defined criteria (1). The remaining 64 patient case reports involved 33 drugs; 5 had level I (definite) evidence, and 13 had level II (probable) evidence. Of these 18 drugs, 5 had not been documented in our previous reviews as causing thrombocytopenia, defined by at least 1 report with level I evidence or 2

Letters
Sarcoidosis Presenting with Massive Involvement of the Nervous System

TO THE EDITOR: Background: Neurologic involvement is a significant cause of morbidity and mortality in patients with sarcoidosis (1).

Objective: To describe a rare case of neurosarcoidosis involving both the central and peripheral nervous systems.

Case Report: A 21-year-old man presented with recurrent fever and weight loss of 1 year’s duration, lower-limb paresthesia, and progressive hyposthenia that had developed in the previous 2 months. Esotropia, diplopia, headache, and vomiting had begun a few days before admission. The patient’s fever had been unsuccessfully treated, and had not responded to several empirical antibiotic therapies. On physical examination, there were signs of multifocal central and peripheral neurologic impairment and mild hepatosplenomegaly. The patient’s temperature was 39 °C. Magnetic resonance imaging showed multiple contrast-enhancing miliary lesions in the brain, cerebellum, brainstem, and spinal cord from the cervical segment to the conus medullaris (Figure). Nerve conduction studies revealed severe axonal sensorimotor neuropathy primarily affecting the lower limbs. Blood tests showed a hemoglobin level of 103 g/L; absolute CD4⁺ and CD8⁺ cell counts of 0.266 × 10⁹ cells/L and 0.160 × 10⁹ cells/L, respectively; and an erythrocyte sedimentation rate of 45 mm/h. Results of tests for HIV-1 were negative. Tests of the cerebrospinal fluid revealed a glucose content of 0.6 mmol/L (11 mg/dL), a protein level of 750 mg/L, and a leukocyte count of 0.076 × 10⁹ cells/L (mostly lymphocytes). Results of cerebrospinal fluid microscopy and cultures for bacteria (including Mycobacterium tuberculosis) and fungi were negative, and oligoclonal banding was not seen. However, results of polymerase chain reaction were positive for M. tuberculosis DNA, and results of a tuberculin test were also positive. Chest radiography was unrevealing. As a result of these findings, therapy with isoniazid, rifampin, ethambutol, and pyrazinamide was started.

After 2 weeks, no significant clinical improvement was seen and cerebrospinal fluid measures were unchanged, except for results of polymerase chain reaction for M. tuberculosis DNA, which were negative. Blood, urine, and stool cultures were also negative for M. tuberculosis. On bone marrow biopsy, we found a noncaseating granuloma, which was negative for acid-fast bacilli on microscopy. After detection of an elevated serum level of angiotensin-converting enzyme, computed tomography of the chest revealed a few small nodular lesions in both lungs and mediastinal lymphadenopathy. A transbronchial lung biopsy showed a noncaseating giant-cell granuloma consistent with sarcoidosis, and bronchoalveolar lavage fluid demonstrated a high proportion of CD4⁺ cells. A gallium-67 lung scan showed a pattern of diffuse uptake. We also performed ophthalmoscopy and found bilateral posterior uveitis. Sarcoidosis was therefore diagnosed, and treatment with methylprednisolone, 60 mg/d, was started. Of the antituberculous drugs, only therapy with isoniazid was continued as prophylaxis. The patient improved gradually, although complete clinical and radiologic remission was achieved after a few months.

Discussion: Clinically evident neurosarcoidosis occurs in 5% to 26% of patients with sarcoidosis (2, 3). It may involve any part of the nervous system, resulting in a wide spectrum of clinical syndromes and yielding a mortality rate of approximately 10%. The lesions may be single or multiple and are usually associated with signs and symptoms of systemic disease. Spinal cord involvement occurs in fewer than 1% of sarcoidosis cases (1, 4). Extended miliary involvement of both the central and peripheral nervous systems is distinctly unusual. The diagnosis of neurosarcoidosis requires the exclusion of other diseases capable of producing a similar clinical, radiologic, and laboratory picture. The management of sarcoidosis is directed at the disease process, rather than the neurologic manifestations, with immunosuppressive agents as the mainstay of treatment. In our patient, the neurologic symptoms were controlled after treatment with isoniazid and methylprednisolone.

Laura Hunt, BA
Xiaoning Li, MS

University of Oklahoma Health Sciences Centers
Oklahoma City, OK 73190

References

Table. Drugs Causing Thrombocytopenia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Individual-patient data</th>
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<tr>
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<td>Simvastatin</td>
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* These 6 drugs were not reported in the previous 3 systematic reviews (1–3) as having evidence supporting a causal relation to thrombocytopenia. For individual-patient data, definite evidence (level I) required reexposure to the drug that resulted in recurrent thrombocytopenia or validation of the causal relation to thrombocytopenia by at least 2 patient case reports with probable evidence (level II), requiring all criteria except reexposure to the drug. For group data, definite evidence (level I) was defined as a significantly increased rate of thrombocytopenia associated with the drug compared with a control group in a randomized clinical trial. Probable evidence (level II) was defined as a significantly increased rate of thrombocytopenia associated with the drug compared with a control group in a nonrandomized study. The complete database of all articles from this report plus our previous reviews, including the definitions of levels of evidence, is available at http://moon.ouhsc.edu/jgeorge.
† One of the level II studies included group data.

Editor’s Note: The lead author of the following Clinical Observation was one of a dozen Associates of the American College of Physicians selected to present a clinical vignette at the 2004 Annual Session in New Orleans. We are proud to present this case report through a special arrangement with the Council of Associates of the College.
histologic picture, above all tuberculosis of the nervous system. The finding of *M. tuberculosis* DNA on polymerase chain reaction in specimens from patients with sarcoidosis (5) does not prove the etiologic role of mycobacteria and may be a misleading diagnostic clue.

**Conclusion:** This report emphasizes the need for greater awareness of the neurologic complications of sarcoidosis. Clinicians should always consider neurosarcoidosis in patients with unusual multiple impairments of the central or peripheral nervous system.

Salvatore Sollima, MD
Francesco Croce, MD
Chiara Scalamogna, MD
Ilaria Caramma, MD
Spinello Antinori, MD
University of Milan, L. Sacco Hospital
20157 Milan, Italy

**References**


**Rapid Diagnostic Test for *Plasmodium falciparum* in 32 Marines Medically Evacuated from Liberia with a Febrile Illness**

TO THE EDITOR: Background: *Plasmodium falciparum* causes 700,000 to 2,7 million deaths annually in the tropics (1) and is the most important infectious risk for civilian and military travelers from developed countries. Diagnostic laboratory resources are often limited in endemic areas, and malaria frequently occurs in large, seasonal outbreaks that can overwhelm expeditious microscopic diagnostic capability. Rapid diagnostic kits have been developed to detect parasite-specific lactate dehydrogenase (the OptiMAL test [Flow Inc., Portland, Oregon]) or histidine-rich protein II (the ParaSight-F test [Becton Dickinson Advanced Diagnostics, Franklin Lakes, New Jersey] and the NOW ICT Malaria P.f./P.v test [Binax, Inc., Portland, Maine]). For the NOW ICT test, sensitivities and specificities for *P. falciparum* infection as high as 100% and 94%, respectively, have been reported (2, 3).

**Objective:** To describe our experience using the NOW ICT test to expedite evaluation of a large outbreak of febrile disease among

**Table. Results of the NOW ICT Test Compared with the Thick-Smear Test***

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<thead>
<tr>
<th>NOW ICT Results</th>
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<td>Negative, n</td>
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* The NOW ICT Test is manufactured by Binax, Inc., Portland, Maine.
Two Patients with Acute Liver Injury Associated with Use of the Herbal Weight-Loss Supplement Hydroxycut

TO THE EDITOR: Background: Many herbal supplements contain compounds that are potentially hepatotoxic (1). Newly formulated Hydroxycut (MuscleTech, Mississauga, Ontario, Canada) is a concoction of plant extracts designed to speed weight loss by increasing metabolism and curbing appetite (2).

Objective: To report 2 cases of severe hepatotoxicity associated with use of the weight-loss aid Hydroxycut.

Case Reports: Two men presented to our emergency department within a 2-month period and were admitted to the hospital. They were previously healthy and reported no recent foreign travel; sick contacts; or risk factors for viral, alcoholic, autoimmune, or hereditary liver disease. Except for Hydroxycut, they reported no recent use of herbal or prescription medications. Both underwent a similar serologic work-up, including viral studies (hepatitis A, B, and C viruses; Epstein–Barr virus; cytomegalovirus), antinuclear and anti–smooth-muscle antibody levels, acetaminophen level, and toxicology screening, which was unremarkable.

Patient 1, a 27-year-old man, presented with 8 days of fatigue and jaundice. He had been taking Hydroxycut for 5 weeks, 3 tablets 3 times per day. Laboratory analysis revealed a serum aspartate aminotransferase level of 1808 U/L (normal range, 5 to 50 U/L), a serum alanine aminotransferase level of 3131 U/L (normal range, 7 to 40 U/L), a bilirubin level of 133 µmol/L (normal range, 0.0 to 1.5 mg/dL), an alkaline phosphatase level of 717 U/L (normal range, 40 to 150 U/L), an albumin level of 39 g/L (normal range, 35 to 50 g/L), a prothrombin time of 16 seconds (normal range, 9 to 13 seconds), and a platelet count of 208 × 10^9 cells/L (normal range, 150 to 400 × 10^9 cells/L). The aminotransferase levels peaked on hospital day 2 (aspartate aminotransferase level, 1969 U/L; serum alanine aminotransferase level, 3962 U/L). Four weeks later, results of the liver function tests had improved substantially (serum aspartate aminotransferase level, 114 U/L; serum alanine aminotransferase level, 304 U/L; bilirubin level, 22 µmol/L [1.3 mg/dL]).

Patient 2, a 30-year-old man, presented with 10 days of jaundice, fever, vomiting, and fatigue. For 5 days, between the 16th and 11th days before presentation, he had been taking 9 tablets of Hydroxycut per day. Except for jaundice and minimal abdominal tenderness, results of physical examination were normal. Laboratory analysis revealed a serum bilirubin level of 133 µmol/L (7.8 mg/dL), an alkaline phosphatase level of 530 U/L, an aspartate aminotransferase level of 59 U/L, a serum alanine aminotransferase level of 45

References
UT, an albumin level of 28 g/L, a prothrombin time of 15 seconds, and a platelet count of 217 × 10⁹ cells/L. An abdominal computed tomography scan and endoscopic retrograde cholangiogram were negative. On hospital day 4, liver biopsy revealed cholestasis and portal inflammation. The laboratory abnormalities improved, and the patient was discharged on hospital day 9. Two months later, results of liver tests were normal.

Discussion: To our knowledge, these are the first reported cases of hepatotoxicity associated with the use of Hydroxycut. Although the evidence reported here is not definitive, the lack of evidence for other causes and the temporal relationship of Hydroxycut ingestion to liver injury suggest a causative relationship. It is not clear which of the ingredients in Hydroxycut may have been responsible for hepatotoxicity (Table). A MEDLINE search did not reveal previous cases of hepatotoxicity resulting from *Garcinia cambogia*, *Gymnema sylvestre*, willow bark, glucomannan, green tea, or guarana extract.

Patient 2 presented with a cholestatic liver injury pattern, and histologic examination confirmed portal inflammation and cholestasis. Several herbs have been reported to produce cholestatic hepatitis including chaparral, kava, and Jin Bu Huan (3). Patient 1 presented with markedly elevated aminotransferase levels; although a biopsy was not obtained, hepatocyte necrosis was the likely pattern of injury. It is not unusual for a single herbal preparation to produce more than 1 type of clinicopathologic liver injury (4).

Conclusion: Evidence for the efficacy of *Garcinia cambogia* in promoting weight loss is not compelling (5). We therefore urge caution in the use of this supplement. Of broader concern are the widespread use of herbal preparations and lack of adequate monitoring of adverse outcomes.

Tyler Stevens, MD
Asif Qadri, MD
Nizar N. Zein, MD
Cleveland Clinic Foundation
Cleveland, OH 44118

References
basal ganglia). In Parkinson disease, this stimulation allows movement of otherwise rigid muscles. Excess stimulation causes excess movement characterized by choreoathetosis.

Because this patient received a water-soluble drug, her blood–brain barrier must have been disrupted. We are left with several hypotheses for the movement disorder: 1) One of the patient’s other medications may have caused increased permeability of the blood–brain barrier. This has not been reported but is something to consider. 2) Trauma occurring during her syncopal episode before admission may have disrupted the blood–brain barrier. 3) The patient may have had a subclinical ischemic event causing increased permeability of the blood–brain barrier. 4) Permeability may have been increased because of another condition, such as multiple sclerosis, tumor, or infection. However, these conditions were ruled out by evidence obtained during the hospital stay. Regardless of the cause of disruption of the patient’s blood–brain barrier, dopamine as a cause of her movement disorder has biological plausibility.

**Conclusion:** In this patient, intravenous dopamine caused acute choreoathetosis, which must have been facilitated by a disrupted blood–brain barrier.

**Victoria A. Walker, MD**
**Maryam Massoumi, MD**
St. Vincent Hospitals and Health Services
Indianapolis, IN 46260

**CORRECTIONS**

**Correction: Office-Based Testing for Fecal Occult Blood**

In an editorial on office-based testing for fecal occult blood (1), the second sentence of the eighth paragraph should have read, “In this survey, 29.1% of the adults 50 years or older who reported having FOBT in the past year said that the only FOBT they received was after a digital rectal examination.”

**Reference**


**Correction: Prolonged Coagulopathy Related to Superwarfarin Overdose**

In a letter on prolonged coagulopathy related to superwarfarin overdose (1), a hemoglobin value was reported incorrectly. The tenth sentence of the Case Report section should have read, “His hemoglobin level had decreased to 79 g/L,” not 7.9 g/L.

**Reference**