Letters to the Editor

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SEIZURE TRIGGERS

To the Editor:

There are increasing reports that video games, a popular form of entertainment among the young, trigger seizures.[1–9] We report the case of a healthy young adult who experienced a generalized tonic-clonic seizure for the first time while playing video games.

A previously healthy graduate student had stayed up late and slept only for 2 hours. The following morning, while playing video games, he experienced a generalized tonic-clonic seizure. He had a history of skull fracture at birth. A computed tomography (CT) scan of the head in 1983 for chronic headache was normal. His father experienced febrile seizures and a maternal uncle has epilepsy. At admission, general physical and neurological examinations and routine laboratory studies were normal. An electroencephalogram (EEG) showed bursts of spike activity in the left parietal area. He has remained free of seizures without antiepileptic drug therapy.

Seizures precipitated by a specific stimulus are classified as reflex epilepsy.[4,8] The most common type is photosensitive epilepsy, in which a visual stimulus triggers an epileptic seizure. Nonvisual stimuli that induce seizures (eg, music, sensory stimuli, reading, eating and mental activity such as arithmetic) are much less common. Photosensitive epilepsy is usually induced by flickering light, such as that produced by television, strobe lights, marquees, and neon signs. The increasing popularity of video games makes them a likely common trigger of photosensitive epilepsy.

Some patients with idiopathic generalized epilepsy are also susceptible to visually induced seizures.[7,9] Others, such as our patient, develop seizures only during photic stimulation. Photosensitive epilepsy usually occurs in children and adolescents. It is almost always generalized tonic-clonic and occasionally absence or myoclonic. Partial seizures are rare. Neurodiagnostic testing is usually normal, but the EEG frequently shows a photoconvulsive response after photic stimulation.

Seizures that occur only after visual stimulation do not require treatment. Avoidance of flickering lights and video games and viewing television from a distance are usually sufficient preventive methods. Children with idiopathic epilepsy are also prone to visually induced seizures and therefore should avoid offending visual stimuli. If photosensitive seizures occur despite these measures, drug treatment with divalproex sodium (Depakote) is usually indicated.

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HERNIA REMISSION

To the Editor:

Direct and indirect inguinal hernias are common and usually require surgical repair. Indirect inguinal hernias are thought to be related to genetic weakness, whereas direct hernias are related to physical strain and tear.[1]

The following case report describes the author: A 39-year-old physician presented with a left inguinal bulge after attempting a marathon run. The patient had neither a personal nor a family history of hernia. On physical examination, the patient had a large left inguinal bulge most notable when standing, present when sitting, and easily reducible when supine. There was no evidence of herniation into the scrotal sac and the external ring appeared intact without evidence of herniation on coughing. The patient saw a family physician and a surgeon; both confirmed the diagnosis and recommended surgery.

The patient elected to use a truss daily for 5 years. The truss kept the hernia reduced and diminished the discomfort. The patient continued to train and participate in several marathon runs while using the truss. Sitting aggravated the discomfort, while the pain abated with extension of the legs. The pain was sometimes aggravated by bowel movement. Sexual relations were not impaired and, in fact, reduced the hernia.

After 5 years, the patient developed a skin lesion on his right ankle. He visited a local podiatrist and immediately had the lesion biopsied. Twelve hours after the surgery, he developed high fever, chills, rigors and infection at the excision site. A wound culture demonstrated *Staphyloccocus* and he was treated with Keflex. The pathologic diagnosis was a benign inclusion cyst. Following this presumed septic episode, the hernia was no longer clinically present.

While asymptomatic direct inguinal hernias may be managed with judicious neglect,[2,3] there has been no documentation in the literature of complete remission of any groin hernia. A direct inguinal hernia is a result of weakening or tearing of the transversalis fascia in Hasselbach's triangle,[2] and does not become scrotal because it does not follow the path of the descending testis. Clinically important, direct inguinal hernias do not strangulate because they are associated with a wide fossa (Hasselbach's triangle) rather than a constricting ring.[2] The incidence of direct inguinal hernia is greatest during the 4th or 5th decade of life. Women appear to be resistant, suggesting a hormonal influence that might make the lower abdominal fascia more pliable. Alternatively, men are more likely to lift heavy things that may result in strain and tear. Familial predisposition is a factor, and defective collagen synthesis in men has been suggested as a cause.[3]

In the present report, closure and remission corresponded with an episode of a lower extremity infection and possible sepsis. I suspect that scarring and adhe-

sions may have occurred at the site of the hernia secondary to lymphangitis. Other speculative opinions are welcome.

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IATROGENIC ALOPECIA

To the Editor:

As our therapeutic opportunities increase and become more complex, so the potential for physicians to cause harm becomes greater.[1,2] This damage may vary from producing death to relatively insignificant symptoms or changes in appearance. These less life-threatening reactions are even less likely to be detected. The following case reports concern two patients with iatrogenic alopecia.

A 70-year-old woman, who had been under my care for a variety of conditions over a period of 31 years, including hypertension and mild angina, reported that she had loss of hair over her entire body during the past several months. At the time she first noted the alopecia, she had been taking propranolol hydrochloride (Inderal) 40 mg three times a day for 8 years and had been applying ½ inch of 2% nitroglycerin ointment to her skin approximately every 8 hours for 4 years.

On review of the possible side effects of her medications, alopecia was noted to be associated with the use of propranolol. Despite the length of time she had been taking this antihypertensive therapy, I decided to change her therapy to methyldopa (Aldomet). Within a month, the patient noticed a gradual regrowth of hair, and within several months, she reported it had reverted to normal. She refused a rechallenge with the former medication.

A 62-year-old woman was placed on 75 mg of imipramine hydrochloride (Tofranil) at bedtime for mild endogenous depression. I had known this patient for over 25 years. Within 1 month of beginning Tofranil, she complained of general hair loss on the scalp. The *Physicians' Desk Reference* (PDR) revealed Tofanil as a possible cause, and she was changed to maprotiline hydrochloride (Ludiomil). Over the next several months, the patient was satisfied that her hair growth had returned to normal. She also refused to resume taking the imipramine.

Because of these experiences, I reviewed a recent edition of the PDR to ascertain the frequency of this side effect and the types of drugs capable of producing it. As is generally well known, antineoplastic agents regularly cause reversible alopecia (35 were listed). In addition, 29 antihypertensive (representing all the various categories), 19 xanthine derivative, 13 nonsteroidal anti-inflammatory, 16 antidepressant, and 7 antiepileptic drugs have produced hair loss. Estrogens also can.

Well over 100 drugs, most of them in frequent use, are capable of producing this adverse reaction. Others include antiarthritic agents of the antimalarial, penicillamine, and gold groups; therapies for gout; anti-Parkinsonism drugs; histamine H_2-receptor antagonists (ulcer medications); lipid-lowering agents; and antiviral medications.

Among the remaining agents known to have produced alopecia, probably the most frequently prescribed are buspirone (BuSpar), diethylpropion hydrochloride (Tenuate), haloperidol decanoate (Haldol), lithium carbonate (Eskalith, Lithonate, Lithone), methimazole (Tapazole), methylphenidate hydrochloride (Ritalin), misoprostol (Cytotec), nitrofurantoin (Macrodantin), selenium sulfide (Selsun) and terfenadine (Seldane).

Except for the antineoplastic agents, most drugs capable of causing hair loss are reported to have an incidence of 1% or less. A few state that this effect may occur as frequently as in 5% of patients using the given medication. While this low incidence may be deceptively reassuring, it should be noted that many of these drugs are utilized for the treatment of various chronic illnesses and therefore are prescribed for extensive periods. Alopecia can begin to develop after a patient has tolerated the drug for an extended duration. It also should be noted that because a patient has exhibited loss of hair from one drug, it does not necessarily follow that the same patient will react adversely to another with apparent chemical similarities. Regrowth of hair can be anticipated when the offending medication is stopped.

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DEPRESSION TREATMENT

To the Editor:

In a recent article on the treatment of depression,[1] the authors discuss the choice of antidepressant medications, emphasizing the new selective serotonin antagonist agents. Seven journal pages are used to describe all of the advantages of these new agents for our patients, despite being told that they are "no more effective than traditional tricyclic antidepressant drugs. . . ." In a section of the article on the "choice of an antidepressant," all the potential advantages of the selective serotonin reuptake inhibitors (SSRI) over traditional agents are highlighted, despite once again stating that they are "no more effective than traditional agents." It is odd that no mention is made of the one major drawback to the use of SSRIs, ie, their cost, in this section, abstract, introduction, or the descriptions of complete profiles of each SSRI. In fact, the cost of these agents is mentioned only once in the last sentence of the article. In contrast, in the following article in the same issue,[2] costs of medications for ulcer treatments are discussed throughout the article as an important variable influencing treatment decisions.

Medication cost is a major factor that physicians should consider and discuss with their patients when prescribing therapies. Such cost decisions can influence compliance with therapy, satisfaction with an encounter, and patient outcomes.[3–5] With the pharmacologic treatment of depression, the cost differentials of commonly prescribed antidepressant agents is substantial (Table). When one of the authors (A.O.G.) first started prescribing the SSRI agents, several angry

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Table. Comparative Costs of Commonly Prescribed Antidepressant Agents

Agent	Dose (mg)	Tablets	Total Daily Dose (mg)	Costs per Month ($)*
Bupropion (Wellbutrin)	75	120	300	68
Fluoxetine (Prozac)	20	30	20	62
Paroxetine (Paxil)	20	30	20	60
Sertraline (Zoloft)	50	30	50	58
Desipramine (Norpramin)†	50	90	150	26
Nortriptyline (Pamelor)†	50	45	75	16
Trazodone (Desyrel)†	100	90	300	14
Amitriptyline (Elavil)†	50	90	150	13

**Cost estimates are based on information obtained by telephone calls to community pharmacies in Chapel Hill, NC.*
†Generically available.

patients telephoned to tell us they could not afford them. Some patients simply showed up 3 months later saying they had quit taking the SSRI agent 2 months before because of the cost. In both circumstances, valuable counseling time was lost, and the patient's care, as well as the physician-patient relationship, was initially compromised.

Currently, we recommend that all patients, including those who are depressed, be informed about the approximate costs of any therapy we plan to prescribe. If there are several agents with equivalent therapeutic profiles, we let them know the benefits (eg, improved adverse effect profile) and the drawbacks (eg, increased costs) of each possible course of therapy. Together, we hope to reach a decision that is best for each patient.

Perhaps the reason that "not all physicians are taking advantage of new developments in effective treatments for depression" is because many patients and clinicians are overwhelmed by the cost of these newer antidepressant agents. Moreover, a recently published meta-analysis comparing tricyclic antidepressants and SSRIs showed no differences in efficacy or acceptability between the agents as first-line treatments for depression.[6] Until therapeutic gains are offered by such agents, a doctrine of informed consent demands that patients and physicians discuss cost considerations as important variables that influence prescribing decisions and subsequent compliance. Authors, journal reviewers, and editors should require cost information as a component of balanced reviews for articles on medications and other medical interventions.

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The preceding letter was referred to Drs Guze and Gitlin, who respond as follows:

We agree with Drs Goldstein and Ives that, for many patients, cost is an important factor in selecting an antidepressant agent. While the data in their table are influenced by regional variations, the trend is the same throughout the country: new agents, especially those still protected by patent, are more expensive than older agents.

However, we feel that the concept of cost must be understood more broadly than a simple description of cost per pill. Especially in this era of evaluating global health care costs, we must consider the *overall* health costs of any treatment. Using this meaning of "cost," the newer antidepressants may be no more expensive than tricyclics, and, in some cases, less expensive. One recent study found that although SSRIs were more than twice as expensive as tricyclics based on medications costs alone, associated costs, such as number of physician visits and decreased number of hospital days were much less, yielding an ultimate *decreased* cost associated with the newer drugs (BCG Study of Midwest Employer Database, personal communication, 1994). Similarly, a recent computer-generated analysis of costs for treating depression in a primary case setting in an HMO found that the overall cost of health care for SSRIs and tricyclics were similar, despite a higher medication price for the newer agents (McFarland B, personal communication, 1994).

Thus, although cost per medication is important, it is less clear that the older medications are truly cost effective when all costs are considered.

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REVERSIBLE GANGRENE

To the Editor:

We would like to report an unusual case and solicit causative suggestions.

A 75-year-old man was in good health and very active until a few weeks prior to presentation. He drove himself to art and aerobic classes and "read constantly and voraciously."

Three weeks before presentation, he complained of pain in his fingers that interfered with his driving. Within 1 week of this, his wife awakened at 4 AM to discover he was missing. The police found him 5 miles from home. He was disoriented and stated that he and his son (who was in another state) were "shoveling snow to get cars out" (it was summer).

The patient's medical history was significant only for hypertension, which was well controlled with enalapril. There was no history of diabetes, Raynaud's phenomenon, or other vascular disorder. He had spent several years in Burma, China, India, and Morocco in the 1940s while in the military. He had no history of ergot ingestion. The patient was a nonsmoker.

On examination, the patient was found to be disoriented and unable to respond appropriately to most questions. Eight of his fingertips were in various stages of dry gangrene. There was no appreciable thickening of the temporal arteries.

Complete blood count chemistries, B_{12}, folate, thyroid panel, complement,

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