liter three years before admission. Instead of providing immediate immune globulin replacement on admission, his clinicians remeasured the serum IgG five days later (130 mg per deciliter), and immune globulin replacement was given seven days after admission. The patient died on the 12th hospital day.

There is an extensive body of literature supporting the value of intravenous immune globulin replacement for patients with CLL with hypogammaglobulinemia and infection.[2-4] The cause of death in this case was determined to be West Nile encephalitis, and case reports suggest that intravenous immune globulin may be therapeutic in such patients without hypogammaglobulinemia.[5] The managing clinicians did not explain the delay in providing replacement, and the discussant did not address the likelihood that immediate immune globulin replacement might have prevented the fatal outcome in this case. Because this was a teaching case, their informed opinions could be valuable.

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THE DISCUSSANT AND COLLEAGUES REPLY: Dr. Newcom suggests that early treatment with intravenous immune globulin might have prevented the fatal outcome of West Nile virus encephalitis in this patient. The patient had received intravenous immune globulin many times over the years because of hypogammaglobulinemia and recurrent bacterial infections, and we agree that it would have been appropriate to give it promptly during the hospitalization in 2003, when infection of some kind was suspected. However, since West Nile virus is a relatively new and uncommon pathogen in the United States, it is unlikely that pooled immune globulin available during 2003 would have had high titers of West Nile virus antibodies. No evidence of West Nile virus infection was detected on a polymerase-chain-reaction assay of the cerebrospinal fluid, so that a search for a preparation with high titers of antibody to West Nile virus would not have been indicated. Although anecdotal reports suggest that treatment with intravenous immune globulin may be of benefit in human West Nile virus infection, no data from controlled trials are available to confirm this. The National Institute of Allergy and Infectious Diseases is currently conducting a placebo-controlled trial in humans infected with West Nile virus of an intravenous immune globulin preparation with high titers of West Nile virus antibodies (ClinicalTrials.gov number, NCT00068055).

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Potential for Abuse of Buprenorphine in Office-Based Treatment of Opioid Dependence

TO THE EDITOR: Buprenorphine was approved by the Food and Drug Administration (FDA) in 2002 for the treatment of opioid addiction in certified physicians' offices. However, the FDA and the Drug Enforcement Administration (DEA) expressed concern that the use of buprenorphine in opioid-dependent populations would inevitably lead to its diversion and abuse.[1,2] Thus, buprenorphine was moved from Schedule V of the Controlled Substances Act to Schedule III.[1] In an effort to restrict the number of persons exposed to the drug, a limit was imposed of no more than 30 patients per qualifying certified physician.

We report on the abuse of buprenorphine products on the basis of data gathered through two well-established networks of several hundred geographically dispersed drug-abuse experts.[3-5] When a suspected case was identified, the drug-abuse ex-

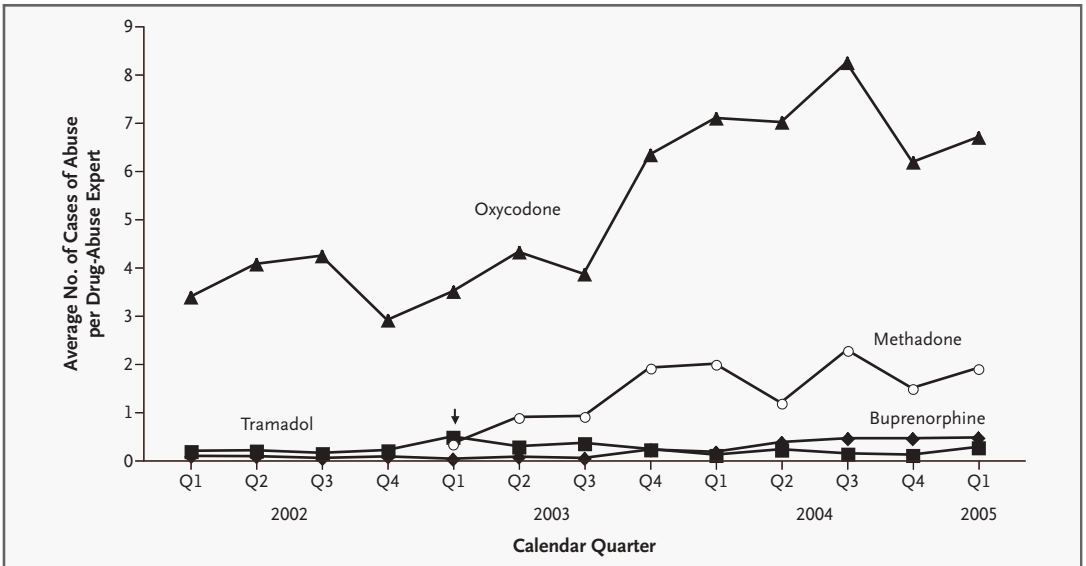

Figure 1. Average Number of Cases of Abuse of Buprenorphine Products, Methadone, Tramadol, and Oxycodone per Drug-Abuse Expert.

The arrow indicates the launch date of buprenorphine for use in office-based treatment of opioid dependence. Q denotes quarter.

perts were asked to complete a structured questionnaire by means of a direct interview with each patient with suspected drug abuse. To place any abuse into perspective, we also assessed abuse of tramadol (an unscheduled drug); methadone (the standard pharmacologic treatment for opioid abuse; Schedule II); and oxycodone (a very widely abused Schedule II opioid analgesic[4]).

Figure 1 shows the average number of case reports of abuse per drug-abuse expert for each calendar quarter of the study period for the drugs examined. Growing abuse of oxycodone was responsible by far for the greatest number, followed by methadone, tramadol, and buprenorphine. There were no statistically significant differences between tramadol abuse and buprenorphine abuse. As reported elsewhere,[3-5] the majority of all prescription-drug abusers were young white men with extensive histories of substance abuse. More than one third of the buprenorphine abusers reported that they took the drug in an effort to self-medicate and ease heroin withdrawal.

These results indicate that there has been very little abuse of buprenorphine since its launch for the treatment of opioid addiction in the first quarter of 2003. The abuse found was no greater than that observed for the unscheduled drug tramadol and much less than that for the Schedule II drugs methadone and oxycodone. There are limitations to this preliminary study: none of our measures correct for the degree of exposure to the drugs in question, since data on exposure are not available. These data could show different rates (cases divided by exposure) from those reported here, but on the basis of the raw number of abuse cases, it would appear that the concern expressed by the FDA and DEA[1,2] about a very large surge in abuse of buprenorphine resulting from its use in an opioid-dependent population may be unfounded, at least during the two years it has been available.

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Damage to Pacemaker Lead during Mammography

TO THE EDITOR: A 73-year-old woman presented on March 22, 2005, for routine evaluation of her VVIR (ventricular pacing, ventricular sensing, inhibition response, rate-adaptive) cardiac pacemaker. The unit had been implanted in the right pectoral region in 1980 because of the sick sinus syndrome. Her current pacemaker lead was the original Cordis lead. On examination, she was alert; she was 1.55 m in height and weighed 40.8 kg. She said that she had no syncope, presyncope, or pectoral wound stimulation.

Investigation of the pulse generator showed that a lead warning had occurred on December 21, 2004, and demonstrated the right ventricular lead impedance to be 3874 ohms (a dramatic increase from 609 ohms on June 10, 2003, and 638 ohms at implantation). A rhythm strip showed frequent failure to sense and total failure to capture by the pacemaker; the patient's native sinus rhythm was normal at a rate of 60 beats per minute. A chest radiograph showed a 1-mm break in the conducting element of the pacemaker lead in the pectoral area. She underwent placement of a new lead and a new pulse generator.

When I saw her in my office five days later, she recalled that she had had screening mammography on December 21, 2004 (the same date that the lead warning had occurred). She recalled that, when her right breast was firmly compressed between the radiography plate and the upper plate, the pain was so excruciating that she screamed, and the procedure was aborted until the pain resolved. Bilateral mammography was then carried out uneventfully. Given the coincident timing of this patient's mammogram and the lead fracture, it is likely that the lead was crushed during the initial attempted mammography. Mammography and pacemaker implantation are both common procedures. More than 30 million women in the United States have mammograms annually; there are also 3 million American women with cardiac pacemakers.[1]

In conclusion, damage to a permanent pacing lead occurred during mammography. During mammographic examination in women with cardiac pacemakers, strict attention must be paid to avoiding damage to the pacemaker.

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