

it is difficult for physicians to critically evaluate the effectiveness of gabapentin for such a wide range of disorders.

We applaud the positive steps outlined by Mr. Peterson and colleagues to reduce commercial influence on educational activities. However, we remain skeptical of the claim that the practices we identified are only of historical interest. Current codes of conduct are largely self-administered and lack an enforcement mechanism, and we know of few systematic data on the extent to which those codes are being followed in letter and in spirit. Similar codes enacted by the pharmaceutical industry, the American Medical Association, and the ACCME, which were in force during the period we studied, were often ignored (1–3). Other research has shown frequent violations of self-regulation by the pharmaceutical industry (4). Thus, we affirm that self-regulation by all parties in these interactions has been insufficient to control undue commercial influence on the practice of medicine.

The risk for such influence persists because of a fundamental conflict of interest, whereby medical education and communications companies and other providers of CME face an incentive to cast a sponsor's products in a favorable light to attract future funding from the sponsor. Despite recent efforts to strengthen guidelines on commercial support of CME, opportunities for abuse still exist (5). Current guidelines allow commercial supporters to raise concerns about content and permit CME providers to consult with commercial supporters about suggested speakers and topics. This creates a condition analogous to that identified by Dr. Sapers as the situation of individual physicians, balancing our ethical and professional obligations against financial interests. However, while physicians and universities that host CME programs have both financial incentives and a fiduciary responsibility to patients and the public, the private, for-profit status of medical education and communications companies raises special concern that financial interests may encroach on the scientific integrity of their educational programs.

Recent strengthening of codes of conduct, stimulated in part by the threat of federal prosecution, has been a welcome improvement in the management of direct and indirect forms of pharmaceutical promotion (6). However, major conflicts of interest and loopholes persist and must be addressed by vigorous regulation with independent oversight to separate commercial from scientific activities.

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CLINICAL OBSERVATIONS

Security Threat Posed by USB-Based Personal Health Records

Background: USB (universal serial bus)-based personal health records enable patients to easily transport their health histories to physicians for review. These small, handheld devices (sometimes called “thumb drives” or “flash drives”) contain a database to store personal health information and a software program to display and edit the contents of the database. They are rapidly gaining popularity (1) and have drawn the attention of the popular press (2) and U.S. Congress (3). Recently, they were distributed to Hurricane Katrina victims in New Orleans as part of the city's Health Recovery Week (4). These devices sell for less than \$100 and are often given free to patients by insurers, employers, hospitals, and health systems.

However, USB-based devices may pose a security threat that could be used to access sensitive data from a physician's computer. By simply inserting the device into a USB port, a provider may put all data on that computer, and potentially all data on the network to which the computer is connected, at risk for theft or corruption.

Objective: To determine whether USB-based personal health records pose a security threat to provider data.

Methods: We identified 5 major USB-based personal health records: the E-HealthKEY (MedicAlert, Turlock, California), Personal HealthKey (CapMed, Newtown, Pennsylvania), Med-Info-Chip (Med-InfoChip LLC, Boynton Beach, Florida), MedKey (MedKey Corp., San Diego, California), and The Bartlett (PEHR Technologies, Salt Lake City, Utah). We obtained 3 of these devices (MedKey Corp. and PEHR Technologies did not supply a sample of their device), analyzed them to determine their structure, and attempted to modify the software program on each device to perform actions of our choosing. No device was manufactured with protections against this.

Findings: We modified the programs on the devices so that, when connected to a computer, they gave the appearance of normal operation but surreptitiously searched for and copied data from the computer to a hidden location on the USB device.

Discussion: The security threat posed by existing patient-controlled USB devices is serious. Depending on how a USB-based personal health record is modified, the programs on the device could tamper with data (for example, to enter unauthorized prescriptions); spread computer viruses; corrupt the hospital or practice network to

which the computer is attached; leave harmful software behind that could, for example, capture usernames and passwords and send them to the person on an ongoing basis; and copy financial or health data—all while the physician is viewing the patient's health record on the device. Each of the devices we reviewed contains a program that must be used to view the patient record, and no reliable mechanism can verify the integrity of these programs. The only certain way for providers to avoid this type of attack is to avoid accepting such devices. Web-based personal health records, which are also available, are a safer alternative. Because they are viewed through a Web browser and require no special software to run, they are not subject to this type of attack.

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Hypothyroidism as a Mimic of Liver Failure in a Patient with Cirrhosis

Background: Hypothyroidism is an unusual cause of ascites. Hypothyroidism may also mimic hepatic encephalopathy in patients with cirrhosis. Reversible, hypothyroidism-induced ascites and encephalopathy simulating liver failure in a patient with cirrhosis awaiting liver transplantation has not been reported.

Objective: To describe the resolution of intractable ascites and encephalopathy with treatment of hypothyroidism in a patient with cirrhosis awaiting transplantation.

Case Report: A 40-year-old woman with cirrhosis secondary to chronic hepatitis C virus infection had intractable ascites and encephalopathy. She was listed for liver transplantation at another institution. She presented for a second opinion to determine whether additional evaluation and treatment might preclude the need for transplantation.

Her medical history included Hodgkin disease that was treated in the 1970s. Chronic hepatitis C virus infection was diagnosed in

1992 and was attributed to blood transfusion. Cirrhosis had been confirmed by liver biopsy. The patient had deteriorated clinically over time despite interferon monotherapy. She had encephalopathy that was unresponsive to lactulose, 120 mL daily, and ascites that did not respond to high-dose diuretics and was managed with large-volume paracentesis. Upper endoscopy showed portal gastropathy without varices. Tests at the referring institution revealed a serum albumin level of 29 g/L (normal range, 34 to 48 g/L), prothrombin time of 13.2 seconds (normal range, 12.3 to 14.6 seconds), alanine aminotransferase level of 60 U/L (normal range, 8.0 to 35.0 U/L), aspartate aminotransferase level of 63 U/L (normal range, 5.0 to 34.0 U/L), and positive hepatitis C virus RNA result.

At presentation, the patient had spider angiomas, ascites, and a mental status examination consistent with stage 3 hepatic encephalopathy. The serum–ascites albumin gradient, which was not calculated at the referring medical center, was 5 g/L, suggesting a high ascites protein content (the serum–ascites albumin gradient in ascites from portal hypertension is usually greater than 11 g/L). In addition, the patient's ankle reflexes, which were tested as part of a routine neurologic evaluation, were markedly delayed in the relaxation phase, strongly suggesting hypothyroidism. These tests and the patient's resistance to lactulose led us to question the diagnosis of liver failure and to test for thyroid function. The patient's thyroid-stimulating hormone level was 155 mU/L (normal range, 0.35 to 5.50 mU/L).

The patient was treated with L-thyroxine, 50 μ g daily increasing to 100 μ g daily. The patient's ascites, encephalopathy, and laboratory abnormalities normalized over 2 months. Liver transplantation was canceled.

Discussion: The patient's clinical course and response to L-thyroxine supplementation suggest that, against the background of hepatitis C virus–induced cirrhosis, hypothyroidism was the cause of encephalopathy and ascites (myxedema ascites). We speculate that the lack of effect of lactulose was attributable to gastrointestinal hypomotility associated with hypothyroidism.

The literature on hypothyroidism simulating liver failure is limited. Yamamoto and colleagues (1) reported a case of cirrhosis with hyperammonemia that presented with dementia due to hypothyroidism. One report of a patient with encephalopathy and hypothalamic hypothyroidism showed improvement in symptoms of hypothyroidism and consciousness disturbance, suggesting that hypothyroidism may aggravate hyperammonemia and encephalopathy (2). In another patient with hepatitis C and hyperammonemic coma who did not clinically improve with encephalopathy treatment, L-thyroxine therapy normalized mental status and hyperammonemia (3). The patient we describe is similar to these cases but may have the atypical feature of a low serum–ascites albumin gradient. The limited literature on serum–ascites albumin gradient in myxedema ascites reports a high gradient (4, 5), the mechanism of which is unknown.

Conclusions: In summary, the case illustrates that hypothyroidism can simulate liver failure in a patient with hepatitis C virus–related cirrhosis. On the basis of this experience and others reported in the medical literature, we believe that clinicians should suspect hypothyroidism and evaluate thyroid function in patients with well-compensated cirrhosis, normal synthetic function as measured by normal prothrombin time, and apparent hepatic encephalopathy that is refractory to treatment.