cal response; most patients improve in this dose range, and drug levels are rarely drawn.

Since toxic concentrations fail to produce reliable clinical signs, drug monitoring may be indicated. More research is needed to elucidate the relevance of slow metabolism and its adverse effects.

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Conn's Syndrome Presenting as Depression

TO THE EDITOR: To our knowledge, Malinow and Lion (1) reported the first case of Conn's syndrome in a 29-year-old woman who appeared with muscle weakness, hypertension, and depression. We report a second case of Conn's syndrome.

Mr. A was a 44-year-old Caucasian man who was seen with a 3-year history of decreased energy, easy fatigability, a sad mood, body aches, decreased concentration, decreased interest in previously pleasurable activities, insomnia, and anxiousness. He had no past or family history of depression and substance abuse. He was diagnosed with hypertension and major depressive disorder. He was treated with hydrochlorothiazide, venlafaxine, and alprazolam in consultation with a psychiatrist. He continued to have poorly controlled hypertension, depression, and hypokalemia despite using increasing doses of hydrochlorothiazide, potassium chloride, metoprolol, ramipril, and venlafaxine. He was referred to our nephrology clinic. Other than systolic and diastolic hypertension, his physical and neurological examinations, including a Mini-Mental State Examination, were normal. His serum sodium level was 139 meq/liter, and his potassium level was 2.1 meq/liter. An examination of his arterial blood gas revealed metabolic alkalosis. His transtubular gradient of potassium was 15 (urine potassium=40 meq/liter, serum osmolality=276 mosmol/kg, urine osmolality=350 mosmol/kg). His plasma aldosterone-to-rennin ratio was greater than 261 (aldosterone=26.1 ng/dl, rennin <0.1 ng/ml). A Doppler ultrasound of his renal arteries was normal. He had a 2-centimeter tumor in his left adrenal gland. His adrenal vein aldosterone levels were markedly elevated on the left side, more so after ACTH stimulation. He was diagnosed with primary hyperaldosteronism and treated with spironolactone, 50 mg b.i.d. He was advised to have laparoscopic resection of his left adrenal gland. His hypertension, hypokalemia, and depression resolved. He stopped taking venlafaxine and alprazolam. He declined to have surgery because he was doing remarkably well 2 months after starting spironolactone.

It is possible that the hypokalemia detected later in this patient may have been the causal factor for his illness. Depression has been reported as a symptom of hypokalemia and other electrolyte disturbances (2, 3). There was no temporal relationship between the start of metoprolol and the onset of depression in this patient. This case highlights the importance of vigilantly considering secondary causes of depression more so in the setting of other medical conditions such as hypertension or electrolyte disturbances.

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Antidepressants and Children's Depression

TO THE EDITOR: In October 2004, the U.S. Food and Drug Administration (FDA) officially announced that it would require manufacturers of the selective serotonin reuptake inhibitor (SSRI) and tricyclic antidepressants to add a black box warning label to each drug's package information (1). This action closes the latest chapter on a decade-long controversy involving SSRIs. I attended both the February and September 2004 FDA hearings on SSRIs and children's depression. During the meetings, I was struck repeatedly by a message I heard from psychiatrists both on the expert panels and those testifying during the open public hearing sections. Again and again I heard them say, "Don't take away the only thing we have to offer these kids."

After the official FDA announcement on the black box warning, the American Psychiatric Association said that it had "deep concern" over the black box warning and its potential "chilling effect" on treatment. "We believe the biggest threat to a depressed child's well-being is to receive no care at all" (2).

Of course, all quotes can be taken out of context, but I'm left feeling disappointed and distressed. First, as a frontline practitioner, I've been asked over and over to practice evidence-based medicine. However, when the evidence in 4,600 children simply isn't there for SSRI effectiveness in pediatric depression, I am now asked to believe, once again, in "professional experience." Even data from the recent Treatment for Adolescents With Depression Study (3), which was meant to bolster the case for fluoxetine in adolescent depression at the hearings, ironically led panel experts to further question the drug's effectiveness and safety (including higher rates of suicidality in the treated versus placebo group).

Most disturbing is the implied role for today's child psychiatrist or behavioral/developmental pediatrician in the treatment of childhood depression. If SSRIs are ineffective for large numbers of youth, have we no other care to offer them? The testimony and response of organized psychiatry to the SSRI controversy suggest just how far the field has moved from the biopsychosocial to the medical model of disease and intervention. A survey of recently trained child psychiatrists (4) found that only one in 10 children in their practices does not receive a medication. I must say that I missed a voice like that of Robert Coles (5) representing the profession at the FDA hearings.

The training, experience, prestige, and power of child psychiatry seem wasted when all of practice is reduced to diagnosis and medication. The role of the child psychiatrist in influencing children, parents, schools, and communities is paramount. Why does it seem that we are more ready to intervene in the environment of the synapse than in the environment of the child? The controversy over SSRIs and children's depression should give us pause to consider "effective" treatments and our own roles in providing them.

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Defining "Health Correlates" in Recreational Gambling

TO THE EDITOR: As a clinician and researcher who is interested in the effects of gambling in later life, I wish to comment on the recent article by Rani A. Desai, Ph.D., M.P.H., et al. (1). The authors examined data collected in 1998 by the National Opinion Research Center. The instrument used was the Gambling Impact and Behavior Study Survey, which is designed to measure gambling behavior and the impact of gambling behavior on selected psychosocial issues. For example, questions on this instrument refer to the number of times an individual has gambled at a casino, how far the person traveled to the casino, how much time was spent there, the type of game played, the amount of money spent, and the amount of money either lost or won at the end of the day. Gambling in lotteries and small business settings and pari-mutuel betting are assessed with similar questions. There are also questions that would indicate problem gambling behavior, such as did you gamble more than you intended? Have you tried to stop, cut down, or control gambling? And are there problems in relationships because of gambling? To further assess the impact, there are questions about legal issues, such as were arguments emotionally harmful? Did an argument ever become physical? And have you ever been arrested?

The abstract of the article states that "recreational gambling in older adults is not associated with negative measures of health and well-being" (p. 1672) when compared with younger adults. This finding is misleading in that Table 2 reports that older adult recreational gamblers have higher rates of past-year alcohol use, abuse, lifetime depression, lifetime incarceration, and lifetime bankruptcy than nongamblers in their age group. Younger adults as well experience higher rates of past-year alcohol use, abuse, lifetime depression, lifetime incarceration, and lifetime bankruptcy than nongamblers in their age group. It appears that gamblers of all ages experience negative measures of health; however, the severity is greater for the younger adult population.

Additionally, there is a limitation that perhaps should have been noted. There is only one question in the 178-question survey that asks respondents to describe their general health. Since "the objective of this study was to identify health and well-being correlates of past-year recreational gambling in adults age 65 years and older, compared to adults age 18–64 years" (p. 1672), a more comprehensive instrument specifically designed to measure general health status could have been employed. The *Handbook of Psychiatric Measures* (2) offers the Short Form-36 Health Survey and the Duke Health Profile as examples. In summary, because of the actual and potential negative consequences associated with recreational gambling among older adults, findings need to be reported cautiously and comprehensively.

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Dr. Desai and Colleagues Reply

TO THE EDITOR: We appreciate Dr. Kerber's thoughtful comments regarding our article on older adult gambling. We share Dr. Kerber's concern that findings should "be reported cautiously and comprehensively" and welcome the opportunity to clarify aspects of our study.

As Dr. Kerber notes, we reported that recreational gambling "is not associated with negative measures of health and well-being." Dr. Kerber asserts that this statement is "misleading," citing findings in Table 2. She states that higher rates of past-year alcohol use and abuse, lifetime depression, lifetime incarceration, and lifetime bankruptcy are seen in the comparison between older adult gamblers and nongamblers. We disagree with her interpretation. Although the absolute proportions of older adult gamblers acknowledging these measures are higher than the corresponding proportions of older adult nongamblers acknowledging them, the between-group differences are not statistically different except for the comparison involving past-year alcohol use. In fact, for past-year alcohol abuse, the adjusted odds ratio is less than 1. Because alcohol consumption can have beneficial effects (e.g., cardioprotection), the finding of an elevated odds ratio between older adult gamblers and nongamblers for past-year alcohol use does not necessarily represent an association between recreational gambling and a negative health measure.

A limitation of the study that we cited in the original report involves the relatively small sample of older adults compared to the larger sample of younger adults. As such, there is less statistical power in the study to detect between-group differences in comparisons of older adult gamblers and nongamblers relative to comparisons of younger adult gamblers and