tients who die while being treated with gatifloxacin for multidrug-resistant tuberculosis might be classified incorrectly and actually die from a severe reaction to the drug. In the light of the study by Park-Wyllie and others, the use of gatifloxacin in patients with multidrug-resistant tuberculosis should be reconsidered.

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THE AUTHORS REPLY: Ittner disputes our suggestion that clinicians should avoid gatifloxacin in favor of antibiotics that do not cause dysglycemia. He cites a retrospective hospital-chart review that grouped cases of hypoglycemia and hyperglycemia together for analytic purposes yet still had insufficient power to detect a difference in risk among the various fluoroquinolones.[1] In contrast, our findings were derived from a population of more than 1 million people, including hundreds who were hospitalized for dysglycemia within days after starting treatment with a fluoroquinolone. Our analysis also incorporated many important determinants of glycemic control, including the use of sulfonylureas and insulin, and adds to a growing body of evidence indicating that gatifloxacin is uniquely associated with dysglycemia.[2,3] We reiterate our suggestion that gatifloxacin be used with extreme caution, if at all, given the widespread availability of alternative antibiotics that do not influence blood glucose levels. In addition, we advise against the overinterpretation of the results of small studies, since such results are particularly prone to a type II error.

The data on mortality rates in our study should be interpreted with caution, because hospitalized patients may have died from infection (rather than from dysglycemia) and because we cannot ascertain the number of outpatient deaths related to dysglycemia. However, we agree with Yadav and Deopujari that the use of gatifloxacin for multidrug-resistant tuberculosis merits reconsideration. Although Bristol-Myers Squibb announced on May 1, 2006, that it would cease marketing its formulation of gatifloxacin (Tequin), other formulations remain available throughout the world, and glucose disturbances during treatment are more likely to have serious clinical consequences in areas where access to medical care is limited.

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Pregnancy in Recipients of Solid-Organ Transplants

TO THE EDITOR: The review by McKay and Josephson (March 23 issue)[1] covers most of the critical issues involved in pregnancy in recipients of solid-organ transplants. However, the rates of infections and their related complications are also high among these patients.[2] Urinary tract infections are the most common bacterial infections and occur in up to 40 percent of pregnant transplant recipients, particularly in patients in whom end-stage renal disease develops after pyelonephritis. It is suggested that pregnant transplant recipients should have monthly screening urine cultures if asymptomatic bacteriuria is present[3] and should be treated for two weeks if an infection is present, followed by suppressive doses of antibiotics for the remainder of the pregnancy. Cytomegalo-

virus (CMV) is the most common viral infection in the post-transplantation period; fetal CMV infection can be diagnosed by culturing amniotic fluid. Herpes simplex virus infection before 20 weeks of gestation is associated with an increased rate of abortion, and a positive cervical culture for the virus at term is an indication for cesarean section. An infant born to a woman who is positive for hepatitis B surface antigen should be given hepatitis B virus immune globulin within 12 hours after birth and receive hepatitis B virus vaccine.

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THE AUTHORS REPLY: We thank Dr. Padhan for highlighting the risk of infections in pregnant recipients of solid-organ transplants. Although we recognize the importance of perinatal infections, space constraints limited what we could cover in the review. Urinary tract infections are common in renal-allograft recipients, whether or not they are pregnant.[1] One reason to recommend pregnancy only after the first post-transplantation year is that the risk of acute CMV infection is decreased[2] and CMV prophylaxis has been completed.

Although hepatitis B virus infection is a worrisome complication, hepatitis C virus (HCV) infection is particularly common in renal-transplant recipients. Fortunately, the incidence appears to be decreasing among patients who receive dialysis, which means that women who receive transplants after dialysis are now less likely to have HCV infection.[3] Vertical transmission occurs in approximately 5 to 10 percent of cases.[4] The likelihood of fetal transmission of HCV may be reduced or eliminated by having transplant recipients plan to become pregnant when their viral load is low or undetectable,[4] data that again provide evidence of the benefit of a planned pregnancy in this population. Another viral infection that should be considered is human immunodeficiency virus (HIV) infection, particularly since HIV-positive patients are now undergoing transplantation more frequently.[5]

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Case 7-2006: A Man with Altered Mental Status and Acute Renal Failure

TO THE EDITOR: As clinical toxicologists, we recommend a few additions and corrections to the causes of anion-gap metabolic acidosis that are listed in the case of ethylene glycol poisoning described by Takayesu et al. (March 9 issue).[1] Propylene glycol, which is used as a diluent in many medications, including lorazepam, is metabolized to lactate, and patients with propylene glycol toxicity can present with elevated anion gaps as well as osmolal gaps.[2] Massive ingestions of analgesics (e.g., acetaminophen and nonsteroidal antiinflammatory drugs) may produce anion-gap acidosis.[3,4] Salicylates, mentioned as a cause of anion-gap metabolic acidosis in both Table 2 and the text of the article, are not cytochrome poisons; they uncouple oxidative phosphorylation by inhibiting an