Lost in Transcription

TO THE EDITOR: In the Clinical Problem-Solving article, “Lost in Transcription” (Oct. 5 issue), Kalus and colleagues quickly home in on a medication error involving methotrexate overdosing as the likely cause of the patient’s fever and presumed urosepsis. However, they do not discuss the fact that this patient, who was reportedly allergic to penicillin, received piperacillin–tazobactam as treatment for her urosepsis — an error that could be more deadly in the immediate future if she did have a true allergy.

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THE AUTHORS REPLY: Harris points out the potential risk of giving a beta-lactam antibiotic to a patient with a documented penicillin allergy. Although our patient did report a history of an allergy to penicillin, we explored this history in more detail before prescribing antibiotics. She described a nonspecific rash after receiving penicillin in the past, but a record review indicated that she had subsequently tolerated several courses of beta-lactams, including piperacillin–tazobactam, without incident. In many patients who report a history of penicillin hypersensitivity, penicillin-specific IgE antibodies are not detected on skin testing. In this case, although we did not have information from skin testing, we considered the patient’s tolerance of piperacillin–tazobactam on previous occasions to be reassuring. No adverse reaction to the beta-lactam was noted on this occasion, despite careful observation for signs and symptoms.

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Reversal of Type 1 Diabetes in Mice

TO THE EDITOR: Dr. Melton (July 6 issue) misrepresents my study on the reversal of type 1 diabetes in mice and its implications for new treatment strategies in humans. As in a previous study, my colleagues and I discovered that an immune therapy triggered a permanent reversal of end-stage type 1 diabetes in mice. The treatment involved two components: injecting the mice with an immune adjuvant (to induce the production of tumor necrosis factor, which destroys autoreactive T cells) and injecting splenocytes from a donor mouse.

Dr. Melton writes that we ascribed the reversal of type 1 diabetes in a mouse model solely to the transplantation of spleen cells and that we claimed to have identified a stem cell among the donor splenocytes that contributed to regrowth of the islets in the recipient. It is true that we observed adult stem cells and that these cells can contribute, in part, to the regrowth of the islets. However, Dr. Melton does not acknowledge that we also observed regeneration of pancreatic islets and complete reversal of type 1 diabetes without the introduction of any live donor splenocytes. Infusion of live splenic cells hastened the development of permanent normoglycemia in the mice but did not enhance the rate of cure. We did not claim that the regenerative process required

Amantadine-Resistant Influenza A (H3N2) Virus in Japan, 2005–2006

TO THE EDITOR: Strains of influenza A (H3N2) virus with a specific mutation (Ser31Asn) have recently shown a dramatic increase in resistance to amantadine in communities in Asia and North America. This resistance in 70 to 90% of strains has occurred despite the absence of sustained selective drug pressure.1,2 We conducted a multicenter study to assess the prevalence of such resistance during the 2005–2006 influenza season in Japan. The study included molecular analysis of the hemagglutinin gene of resistant and sensitive influenza A (H3N2) viruses.

We examined a total of 415 isolates of influenza A virus, sampled from November 2005 to April 2006, for amantadine resistance. Of these samples, 231 of 354 influenza A (H3N2) viral isolates (65.3%) were amantadine resistant, with a Ser31Asn change in the M2 gene. The prevalence of resistance ranged from 36.8 to 100%, according to the area in Japan. However, none of 61 influenza A (H1N1) viral isolates were resistant. Analyses of the hemagglutinin gene in influenza A (H3N2) viral isolates showed two distinct clades: all amantadine-resistant viruses were in clade N, and all amantadine-sensitive viruses were in clade S.

The clinical presentation did not differ between patients shedding clade N virus and those shedding clade S virus. None of the patients had received previous treatment with amantadine. The numbers of patients with influenza at the medical facilities participating in the study were similar to those in the past, despite a high proportion of resistance. Clade N viruses were also found in other countries, suggesting that this strain predominated not only in Japan but also in other