and Drug Administration.” We also said, “High-dose interleukin-2 induces responses in 21 percent of patients, as compared with only 13 percent of patients who receive low-dose interleukin-2.” We could have cited the McDermott study as well.

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To the Editor:

Baker and Cannon (Nov. 3 issue) describe a patient with left-sided ptosis and mydriasis after cranial trauma. The computed tomographic scan in Panel C of the image shows a bone fragment postero medial to the dorsum sellae, suggesting a fracture of the anterior part of the petrous bone, rather than a lesion of the sella turcica, which is located more medially and cranially and would not be visible on the slice presented. A fracture of the anterior petrous bone may affect the third cranial (oculomotor) nerve as well as the eighth (acoustic) nerve. This matches perfectly with the clinical symptoms presented — that is, mydriasis (parasympathetic portion of the third nerve) and ptosis (motor portion of the third nerve), together with tinnitus and vertigo (eighth nerve). The combination of these symptoms is improbable in a patient with a fracture of the sella turcica. A careful clinical examination including pupil reactions and ocular movements would have been extremely helpful to localize the traumatic lesion.

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The authors reply:

Drs. Cruysberg and Theelen have raised some interesting points. Their disagreement with our description of the fracture may be related to a difference in terminology. In fact, the sella turcica is a fossa within the sphenoid bone, which is fractured in the image in the report. Perhaps it would have been better to describe the lesion as a complex fracture of the lateral aspect of the sphenoid bone. Our neuroradiologists do not agree that the image shows a fracture of the anterior petrous bone.

Finally, with regard to performing a careful clinical examination, we would point out that the patient was intubated and paralyzed on arrival at the trauma center. The appropriate examinations were performed when the patient stabilized and was extubated.

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To the Editor:

In the discussion of the clinical syndromes of mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (the MELAS syndrome), Dickerson et al. (Nov. 24 issue) do not mention L-arginine therapy. This option should be considered in the treatment of patients with stroke-like episodes. Although no randomized trial of L-arginine therapy for the MELAS syndrome has been performed, observational data support its use, particularly in the acute phase of the disorder. Improvement has been reported in stroke-like symptoms within 24 hours after the use of L-arginine therapy.