shown to be WG by lung biopsy. Our patient also had striking meningeal and cerebral involvement. The subacute meningitis and brainstem parenchymal disease preceded multisystem disease, including bilateral chorioretinitis, by several months. A biopsy of the meninges was not done, so we could only assume that his brainstem and meningeal disease was due to WG. After consultation with Dr. Anthony Fauci at the NIH, this patient was treated with cyclophosphamide/prednisone and made an excellent clinical recovery. We believe our report is the first showing reversal of meningeal disease in WG with immunosuppressive therapy.

Our patient has had no recurrence of his granulomatous disease although he has persistent mild neurologic findings and is blind in one eye. He ultimately developed a seizure disorder, but sophisticated imaging studies, not available in the mid-1970s, revealed no new disease.

Gage Van Horn, MD
Houston, TX

Reply from the Authors: We thank Dr. Van Horn for drawing our attention to his and Dr. Atcheson's case report. We agree that although the diagnosis was not biopsy-proven, their patient's meningeal and cerebral involvement was due to WG. Taken together, these two reports emphasize the beneficial effect that immunosuppressive therapy may have on this form of WG.

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Cyclosporine and Duchenne muscular dystrophy

To the Editor: Sharma et al¹ reported improvement of clinical function in children receiving cyclosporine therapy for 8 weeks. The study was prompted by the beneficial effect of prednisone,² which reduced the inflammation in muscle, decreasing the number of cytotoxic lymphocytes in children with Duchenne muscular dystrophy.³

With the same aim, we performed an 8-week trial in 20 male dystrophic hamsters (Canadian Hybrid Farms, Centerville, NS, Canada; CHF-146). Ten animals received cyclosporine A (provided by Sandoz Pharmaceuticals, East Hanover, NJ) by subcutaneous injection in the neck at a dose of 1.2 mg/kg/d. Ten control animals received injections of normal saline solution. Seven hamsters that received cyclosporine and nine that received saline survived. Hamsters were anesthetized by an overdose of ketamine/xylazine and aortic blood was taken. Serum CK activity and a blind histologic evaluation of the quadriceps femoris and ventricle were done, using previously reported techniques.⁴

No difference in histologic parameters was detected in the two populations of animals. However, serum CK was significantly increased in the animals receiving cyclosporine, with levels of $1,856 \pm 208$ U/l (mean $\pm$ SE) compared with $1,092 \pm 231$ U/l in the control animals.

The elevation of serum CK might suggest a possible adverse effect of cyclosporine in these animals, although this was not visible in the histologic studies. The possibility that cyclosporine might have aggravated the cardiomyopathy, which these animals have in addition to skeletal muscular dystrophy, may have to be considered. We did not measure CK isoenzymes to demonstrate whether this came from cardiac or skeletal muscle.

It has been shown that cyclosporine can inhibit myoblast fusion and alter calcium homeostasis^{5,6} and that it has been associated with rhabdomyolysis⁷ and myopathy⁸ in humans. These and our observations should be taken under consideration in the

future, particularly in therapeutic trials or in the use of immunosuppression in myoblast implant studies in muscular dystrophy.

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Linda H. Horner, HT(ASCP)
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Reply from the Authors: Bertorini et al report that cyclosporine administration to dystrophic hamsters produced an elevated serum CK, which is interesting but nonspecific. There were no histologic changes associated with cyclosporine treatment, and we are not told whether baseline CK measures were different in the two groups nor whether there were any clinical differences following cyclosporine. Many factors influence the serum CK, and neuromuscular clinicians have learned to be cautious about interpreting the diverse changes in serum CK in response to different drugs.

Our study was designed only to measure the bottom line of muscle performance, namely altered force generation in patients with Duchenne muscular dystrophy following cyclosporine treatment. The improved force generation was found not only in our initial 2-month study¹ but also during our longer trial⁹ (6 months) of myoblast transfer, which demonstrated a longer-lasting beneficial effect of cyclosporine on muscular force production in Duchenne muscular dystrophy.

It is true that rare cases of myopathy have been associated with cyclosporine,^{7,8} but dramatic improvement in patients with myositis has also been described with cyclosporine treatment.^{10,11} Nonetheless, we agree that cyclosporine must be used with considerable caution and must be carefully monitored in any clinical trial of patients with muscular dystrophy.

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Correction

In "Evidence of allelic imbalance of chromosome 6 in human astrocytomas," by Liang et al, which appeared in the March issue (*Neurology* 1994;44:533-536), there is an error in figure 1. At the locus D6S29, the B1 allele size should be 13 kb, not 1.3 kb.

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Correction

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