generates the question: Would this high exacerbation rate happen if patients were allowed to achieve remission with steroids without using MMF?

It was previously illustrated that among patients with MG who were treated with steroids, 80.2% (93/116) achieved remission/minimal manifestations status and only 18% (17/93) experienced an exacerbation afterward.[2] This clues in that the high exacerbation rate reported by Oskarsson et al. after discontinuing MMF may not reflect the natural disease activity but rather reflects MMF dependence, and that continuing MMF mainly treats the dependence. Both studies are limited by a lack of information on the prednisone dose required to maintain remission/minimal manifestations status (an important data point to determine whether MMF is effective).

Author Response: Björn Oskarsson, Sacramento; David M. Rocke, Davis; Karsten Dengel, Sacramento; David P. Richman, Davis, CA: We thank Dimachkie et al. for their interest in our article.[1] However, in opposition to Dimachkie et al., we would consider MG exacerbations after discontinuation of MMF as MG exacerbations rather than a novel MMF dependence condition. Given this premise, patients with pharmacologically controlled MG seem a more appropriate control group compared to patients who had MG but sustain remission without pharmacologic treatment. We would suspect that patients without symptoms or treatment may have a less-active disease compared to a population requiring treatment. This last group is also rare in our clinic, further making such a comparison less meaningful.

The patients were selected on the basis of being on stable doses of prednisone (0–25 mg/d; see table 2)[1] and only MMF was varied. Our article does not address corticosteroid treatment of MG and we do not argue that corticosteroids are not an effective treatment of MG. The interest in treating MG with MMF stems primarily from MMF's more favorable side-effect profile.

1. Oskarsson B, Rocke DM, Dengel K, Richman DP. Myasthenia gravis exacerbation after discontinuing mycophenolate: a single-center cohort study. Neurology 2016;86:1159–1163.
2. Pascuzzi RM, Coslett HB, Johns TR. Long-term corticosteroid treatment of myasthenia gravis: report of 116 patients. Ann Neurol 1984;15:291–298.

CORRECTIONS

Neurofilament light chain level is a weak risk factor for the development of MS

In the article "Neurofilament light chain level is a weak risk factor for the development of MS" by G. Arrambide et al.,[1] there is an error in figure 2. In panel C, the p value should read "$p < 0.0001$" rather than "p , 0.0001" as originally published. The editorial staff regrets the error.

REFERENCE

1. Arrambide G, Espejo C, Eixarch H, et al. Neurofilament light chain level is a weak risk factor for the development of MS. Neurology 2016;87:1076–1084.

Pediatric multiple sclerosis: Conventional first-line treatment and general management

In the article "Pediatric multiple sclerosis: Conventional first-line treatment and general management" by A. Ghezzi et al.,[1] there is an error in the fourth author's name, which should have read "Teri Schreiner" rather than "Teri Shreiner" as originally published. The authors regret the error.

REFERENCE

1. Ghezzi A, Amato MP, Makhani N, et al. Pediatric multiple sclerosis: conventional first-line treatment and general management. Neurology 2016;87(suppl 2):S97–S102.

Author disclosures are available upon request (journal@neurology.org).