Delayed facial palsy after microvascular decompression for hemifacial spasm

TO THE EDITOR: We read with great interest the article by Lee et al.3 (Lee JM, Park HR, Choi YD, et al: Delayed facial palsy after microvascular decompression for hemifacial spasm: friend or foe? J Neurosurg [epub ahead of print September 1, 2017. DOI: 10.3171/2017.3.JNS162869]). The authors reported that 45 of 310 patients (14.5%) with hemifacial spasm (HFS) developed delayed facial palsy (DFP) after microvascular decompression (MVD). At final follow-up, 44 patients (97.8%) completely recovered after corticosteroid treatment. In addition, they found that patients who experienced an immediate disappearance of spasm after MVD had a higher risk of DFP, and they also found that preoperative botulinum neurotoxin injections negatively influenced the occurrence of DFP. We commend the authors for their study, which is a valuable contribution to our understanding of DFP after MVD in patients with HFS. However, we hold that several controversies still exist in DFP that should be further discussed.

Although facial nerve weakness following MVD is unusual, when it happens, it usually has a negative effect on patients’ quality of life, increasing their psychological pressure and causing them to question the efficacy of the operation. Generally, the facial palsy can be divided into two types, immediate facial palsy (IFP) and DFP, based on when it occurs. IFP occurs at once after the operation, and it is mainly due to the intraoperative damage to the facial nerve. However, there is no consensus definition of DFP. In order to differentiate DFP from IFP, most authors have used the criterion that the facial palsy occurs over a 24-hour period after the operation, just as the authors of the present study.

Notably, the incidence of DFP following MVD in the present study (14.5%) was close to twice as high as that reported in previous studies.2,4–6 The authors speculated that the higher rate of DFP observed at their institution might result from the stimulation of the facial nerve by the moved long-shape Teflon felts. This hypothesis might be true or at least plausible. Nevertheless, we wonder why they insert at least 3 Teflon felts in every patient. In our opinion, the number of Teflon felts should be moderate, as too many Teflon felts might lead to a higher risk of nerve compression. Furthermore, the authors found that a high House-Brackmann (HB) grade correlated with late onset of DFP, but the recovery time from the onset of DFP was not related to the HB grade. These results are interesting but seem a little different from what might be more common sense—that is, that patients with higher grade of facial palsy might require longer recovery periods. In a similar study performed by our team earlier,2 we demonstrated that the time of onset was correlated with the duration of DFP; earlier development of DFP corresponded with a shorter duration, whereas later development of DFP corresponded with a longer duration. Unfortunately, we didn’t analyze the correlation between the onset time and the HB grade of DFP in that study.

In addition, in their study, patients with DFP after MVD were treated with oral or intravenous steroid therapy. However, the best method of treatment for patients with DFP remains debated because the pathophysiological mechanism of DFP is unclear, and it is also puzzling why DFP usually occurs up to several days after surgery. Evidence suggests that multiple factors, acting in isolation or as collaborating mechanisms, may underlie the pathophysiology. These factors include viral reactivation, facial nerve exit zone injury via the Teflon felt, local perineural edema, and vasospasm.1,4,6 Hence, the treatments for DFP are all empirical therapies. Fortunately, however, the prognosis of DFP is favorable, with almost all reported patients recovering normal or near-normal facial function within a few months.2,4–6 In our prior study,2 most patients with DFP recovered spontaneously, although without any treatment. Therefore, it is still unclear whether treatment is necessary for patients with DFP. In addition, further large-sample controlled studies are obviously needed for evaluating how the effects of treatment such as corticosteroid administration or antiviral medication would influence the length of recovery when DFP occurs.

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References
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Response

We are grateful to Dr. Lei and colleagues for their detailed and thoughtful letter regarding our article. Our goal was to report that the disappearance of spasms immediately after MVD was the only prognostic indicator of DFP.

Dr. Lei correctly points out 3 important issues. First, should we insert at least 3 Teflon felts in every patient? (Too many Teflon felts may lead to a higher risk of nerve compression.) Second, was the recovery time from the onset of DFP related to the HB grade of DFP? (This article demonstrated that there is no relation to HB grade.) Third, even though the method of treatment for DFP remains controversial and the prognosis of DFP is favorable, does one have to use steroid for treatment of DFP?

First, as the authors have pointed out, we agree that the number of Teflon felts can have an effect. However, Teflon felt is not standardized in size, length, and shape, and there is no objective comparison of the size, shape, and number of Teflon felts used according to the surgeon. Since the thickness, length, and shape of the Teflon used by each surgeon differ, the number of Teflon felts may not be an objective parameter. Additionally, it is difficult to generate enough space with one or two pieces of the felt because we use thin Teflon felt to enable easier handling. Therefore, we made space by using more than 3 pieces. The effect of thickness, length, shape, and number of Teflon felts is also likely to be worthy of objectification.

Second, if DFP were caused by a mechanical injury, it is obvious that the degree of severity would affect the recovery period. The exact cause of DFP has not been determined to date. Since it is known that the cause is not a mechanical injury, it is presumed that it will not be correlated with the recovery period. Naturally, there may also be cases in which an author finds a correlation with a previously published article. Accordingly, more studies of this issue are required.

Finally, treatment of DFP is not standardized. Patients are worried about a full recovery, as mentioned earlier. Although DFP is said to be self-limiting from the point of treatment, we hope for a more rapid recovery. Among the estimated causes of DFP, steroid therapy is preferred in terms of treatment in cases of edema and immune factors for its effectiveness. Accordingly, our hospital has generally used steroids as a protocol for the treatment of DFP. However, in our opinion, a study is needed to compare the effects and necessity of steroids because we did not analyze the effects of treatment on DFP in this study.

Many future studies should refine treatment and improve results in the future. Once again, we appreciate the response of Dr. Lei and colleagues, and these ongoing debates should help our community make HFS treatment an increasingly effective and safe endeavor.

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Utility of folate receptor–targeted fluorescent dye for resection of pituitary adenomas

TO THE EDITOR: I read with keen interest the article by Lee et al. (Lee JYK, Cho SS, Zeh R, et al.: Folate receptor overexpression can be visualized in real time during pituitary adenoma endoscopic transsphenoidal surgery with near-infrared imaging. J Neurosurg [epub ahead of print August 25, 2017. DOI: 10.3171/2017.2.JNS163191]). The technique of intraoperative pituitary tumor visualization using a folate analog conjugated to a near-infrared (NIR) fluorescent dye is a promising contribution as an adjunct technique in the surgeon’s armamentarium. In their limited series of 3 patients with folate receptor alpha (FRα) overexpression, intraoperative fluorescence perfectly predicted the postoperative MRI findings. Based on