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Sir,
Occurrence and severity of upper eyelid skin contracture in facial nerve palsy

In Occurrence and severity of upper eyelid skin contracture in facial nerve palsy,[1] Ziahosseini *et al* quantitatively demonstrates a shortening of the upper eyelid by a standardized measurement between the upper eyelid margin and the lower border of brow in 66 patients with facial nerve palsy. They explain these findings, seen in 71% of their patients, with contractions of the upper eyelid skin: local atrophy of muscle, and/or soft tissue could result from disuse or diminished activity caused by the palsy. Furthermore, muscle-pump paralysis could also reduce venous tone and therefore may raise the hydrostatic pressure within tissue.[2] Following their argumentation, these could lead to fibrosis of the subcutis and trophic skin changes. Focusing on ocular problem in patients with facial paralysis, Ziahosseini *et al* not only highlight this till now often ignored problem, but also introduce an easy way to implement quantitative measurement for this specific change of the upper eyelid. The authors hope that in future studies the etiology and the pathophysiology of this phenomenon could be clarified by examine patients with complete transection of the facial nerve. These patients should develop a more severe contracture because of the lack of any muscle activity. Also a longitudinal study could show the development of these shortening of the upper eyelid in such patients.

We strongly encourage the authors to start these studies. Two diagnostic tools, successfully used in patients with facial paralysis by us, could provide additional information: electromyography, especially needle electromyography, could verify and also quantify the muscle activity on the 'paretic' side. To our experience, the majority of patients initially suffering under a facial palsy will end with a misdirected reinnervation of the mimetic muscles. Synkinesis and mass movements are often seen in this chronic patients starting with 3 months after the lesion.[3] The weak negative correlation between the SFGS subscore for synkinesis and the upper eyelid skin shrinkage suggests that reinnervation could reduce the problem. But the other way round, strong ocular hyperinnervaton could also explain the eyelid skin shrinkage by a constant contraction of the orbicularis oculi muscle. Electromyography would be an ideal tool to test both hypotheses. The second tool would be a high frequency ultrasound of the ocular skin and the muscle.[4] The diameter of the orbicularis oculi (Figure 1) would tell us if it is a denervated muscle with atrophy or a misdirected reinnervated one were you often see an increased diameter compared with the non-paretic site.[5] In addition, the skin itself and the subdermal connection tissue could be directly visualized by high frequency ultrasound. Again, a comparison with the non-paretic site should help to understand what is going on in the weeks and month after a facial paralysis: are there really chronic edemas and contractions of the skin, or can much of the

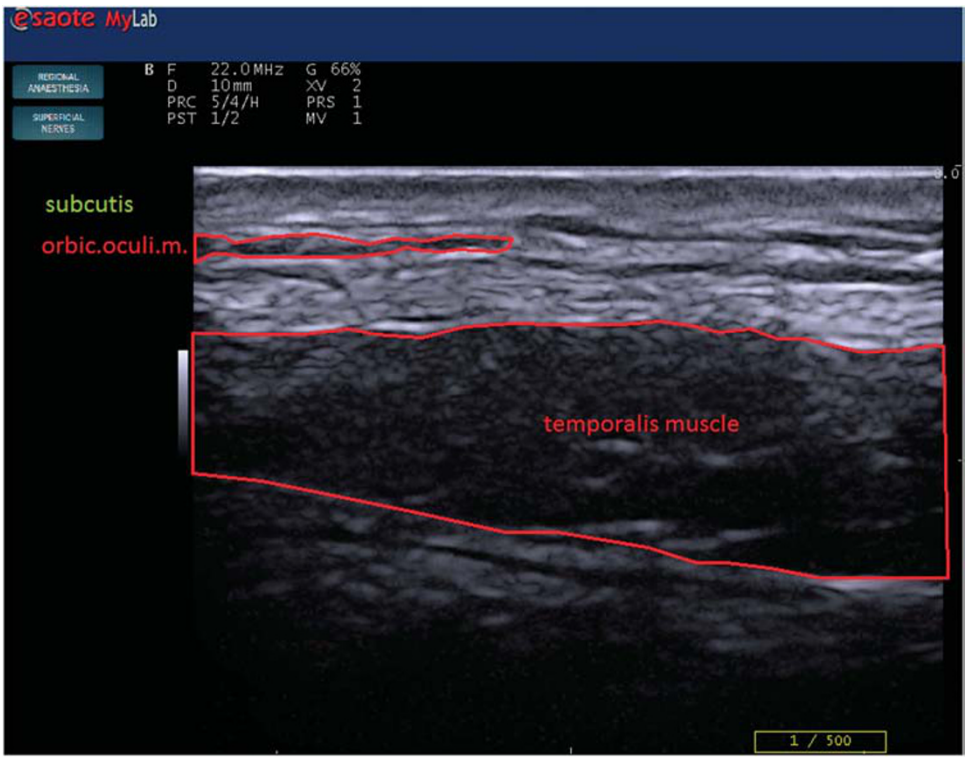

Figure 1 High-resolution ultrasound image showing the lateral part of the orbicularis oculi muscle and the medial part of the temporalis muscle. The 22 Mhz ultrasound transducer is placed in an axial plane 5 mm lateral of the lateral corner of the left eye. Total depth of the image is 10 mm. The red lines represent the borders of the muscles.

phenomena be explained by the increased permanent muscle activity?

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Neovascular age-related macular degeneration: is it worthwhile treating an eye with poor visual acuity, if the visual acuity of the fellow eye is good?

Age-related macular degeneration (AMD) is a bilateral disease and the incidence of neovascularisation (nAMD) in the fellow eye is about 20–42% in the first 2–3 years.[1,2] Many patients have a very different visual acuity (VA) in the two eyes at the first visit. Treatment of the first eye

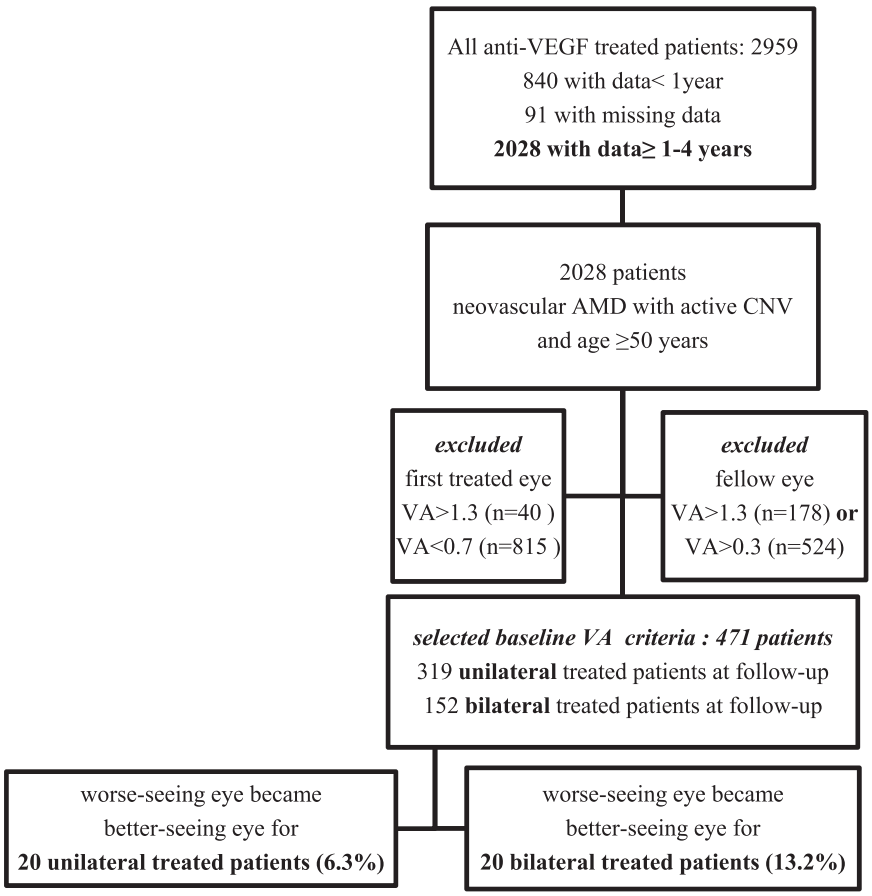

Figure 1 Patients with neovascular AMD in a treatment protocol with intravitreal ranibizumab or aflibercept injections in a pro re nata regimen. Included patients fulfil a selected criteria in visual acuity (LogMAR), first-treated eye, visual acuity ≥ 0.7, and fellow eye visual acuity ≤ 0.3 and the patients have more than 1 year of follow-up.