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CORRESPONDENCE

Comment on: A trial of a mechanical device for the treatment of blepharospasm

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To the Editor:

Fantato et al. reports a mechanical methodology for blepharospasm treatment [1]. We report a case of Apraxia of Eyelid Opening (AEO) treated with apraclonidine, the first case of its kind in the English ophthalmic literature, which could indicate a pharmacological alternative.

A 72-year-old, Caucasian male with benign essential blepharospasm presented with a 1-year history of bilateral, simultaneous, passive ptosis and impaired voluntary eye opening. His eyes would stay open if manually opened or if he tapped his check. Botulinum toxin injections improved symptoms, but the effect dissipated after several weeks. There was no evidence of myasthenia gravis, including fatigability or orbicularis weakness, neurogenic ptosis, or third nerve palsy.

His medical history was significant for obstructive sleep apnoea, coronary artery disease, and peptic ulcer disease. Surgical history included cataract extraction in both eyes (OU), LASIK, chronic dry eye, and blepharoplasty OU. Medications, family history, and systems review was non-contributory. Examination revealed best corrected visual acuity of 20/20-2 in the right (OD) and 20/20 in the left (OS). Pupils measured 4 mm in dark and 2 mm in light OU, with no relative afferent pupillary defects. External exam showed difficulty with initiation of eyelid opening OU (Fig. 1a). Intraocular pressure was 16 mm Hg OU. Ishihara colour plates were 14/14 OU. Slit lamp and fundus examination were non-contributory.

One drop of apraclonidine 0.5% was applied OU, with improvement was noted OU (Fig. 1b). No adverse effects have been reported, and long-term efficacy will be evaluated at the next appointment.

The pathophysiology of AEO, a non-paralytic, non-blepharospasm related, motor disorder that inhibits voluntary eyelid opening, may involve supranuclear levator palpebrae muscle inhibition [2], which can be counter-inhibited by botulism therapy [3]. Apraclonidine, a selective α2 receptor with weak α1 affinity, may stimulate α1 receptors in the levator muscle, overriding the overactive

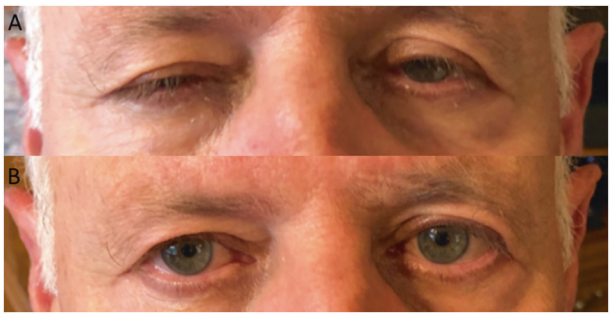

Fig. 1 **a** External exam showed difficulty with initiation of eyelid opening in both eyes (OU). **b** One drop of apraclonidine 0.5% was applied OU, with improvement was noted OU

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pretarsal orbicularis activity responsible for AEO [4]. We propose apraclonidine stimulation of the inhibited levator muscle to reverse of AEO, comparable to reported apraclonidine treatment for ptosis [5].

Further study is needed to validate this treatment. However, if a trial of apraclonidine produces improvement while in clinic, we suggest apraclonidine could be offered as therapy.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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