Thixotropy of levator palpebrae as the cause of lagophthalmos after peripheral facial nerve palsy

Aramideh, M.; Koelman, J.H.T.M.; Devriese, P.P.E.O.M.; Speelman, J.D.

Published in:
Journal of Neurology, Neurosurgery and Psychiatry

DOI:
10.1136/jnnp.72.5.665

Citation for published version (APA):
Thixotropy of levator palpebrae as the cause of lagophthalmos after peripheral facial nerve palsy

M Aramideh, J H T M Koelman, P P Devriese, J D Speelman and B W Ongerboer de Visser

doi:10.1136/jnnp.72.5.665

Updated information and services can be found at:
http://jnnp.bmjjournals.com/cgi/content/full/72/5/665

These include:

References
This article cites 10 articles, 5 of which can be accessed free at:
http://jnnp.bmjjournals.com/cgi/content/full/72/5/665#BIBL

1 online articles that cite this article can be accessed at:
http://jnnp.bmjjournals.com/cgi/content/full/72/5/665#otherarticles

Rapid responses
You can respond to this article at:
http://jnnp.bmjjournals.com/cgi/eletter-submit/72/5/665

Email alerting service
Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

Topic collections
Articles on similar topics can be found in the following collections

Neuromuscular disease (353 articles)

Notes

To order reprints of this article go to:
http://www.bmjjournals.com/cgi/reprintform

To subscribe to Journal of Neurology, Neurosurgery, and Psychiatry go to:
http://www.bmjjournals.com/subscriptions/
Thixotropy of levator palpebrae as the cause of lagophthalmos after peripheral facial nerve palsy

M Aramideh, J H T M Koelman, P P Devriese, J D Speelman, B W Ongerboer de Visser

Peripheral facial nerve palsy is the most commonly encountered cranial nerve disorder, with an annual incidence of 35 per 100 000.1,2 It has various causes, and there is an association between Bell’s palsy—the “idiopathic” form of facial nerve palsy—and hypertension and diabetes mellitus.3

The functional morbidity and aesthetic abnormalities associated with facial nerve palsy mean that early diagnosis and rapid treatment are required. Prevention of corneal exposure and ulceration caused by lagophthalmos—that is, the incomplete closure of the affected eyelid—is specially important. Many types of treatment have been developed to prevent or to minimise the complications of lagophthalmos after facial nerve palsy.4,5

The orbicularis oculi muscle (the eyelid closer) and the levator palpebrae muscle (the eyelid opener) act antagonistically during various eyelid movements. During a blink or voluntary forceful closure of the eyelids, the levator palpebrae relaxes, releasing the passive, elastic downward forces stored in the eyelid ligaments, while the orbicularis oculi muscle contracts, causing rapid lowering of the eyelid. As soon as the orbicularis oculi activity returns to its quiescent state, the levator palpebrae resumes its tonic activity, raising the eyelid to its previous position.6,7

At first sight, it seems logical to assume that lagophthalmos is directly caused by paresis of the orbicularis oculi muscle. However, there are several reasons to believe that this is not the case. First, it is known that gentle closure of the eyelids, as occurs during sleep, is not caused by contraction of the orbicularis oculi, but is brought about solely by inhibition of the levator palpebrae muscle. However, in patients with facial nerve palsy the affected eyelid remains open during sleep. Second, during downward saccadic movement of the eyes, there is associated downward motion of the upper eyelids and a smooth pursuing downward movement of the eyelid following downward movement of the eyes. These upper eyelid movements are mainly caused by gradual inhibition of levator palpebrae activity.8 However, it is our observation that patients with facial nerve palsy and lagophthalmos seem unable to perform these movements properly on the side of paresis.

In the light of these findings, we hypothesised that the lagophthalmos in such patients might reflect thixotropy of the levator palpebrae muscle. Thixotropy is stiffness of a striated muscle because of the formation of tight crossbridges between the actin and myosin filaments within muscle fibres. This thixotropic response might occur because of the disturbed blinking on the side of the facial nerve paresis, the levator muscle not being stretched sufficiently to detach the crossbridges. We have tested this hypothesis by manually stretching the levator palpebrae muscle and measuring the alteration in lagophthalmos.

METHODS

In a prospective open study, we examined 13 patients with a facial nerve palsy, nine male and four female, aged between 17 and 73 years (mean 38 years). Nine had Bell’s palsy and the others had facial nerve palsy from various causes. The time interval between the occurrence of facial nerve palsy and our investigation varied from one day to six months (median 18 days). All patients had lagophthalmos of varying degree. The patients were asked to close their eyelids gently, as though they were attempting to sleep, at which point we measured the width of the palpebral fissure and hence the amount of lagophthalmos.

Our intervention consisted of complete passive closure of the affected eyelid, followed by manual stretching of the upper eyelid, and hence of the levator palpebrae muscle. Thixotropy might occur because of the disturbed blinking on the side of the facial nerve paresis, the levator muscle not being stretched sufficiently to detach the crossbridges. We have tested this hypothesis by manually stretching the levator palpebrae muscle and measuring the alteration in lagophthalmos.

Figure 1 The width of the palpebral fissure measured before and after the upper eyelid stretch manoeuvre in 12 patients with peripheral facial nerve palsy. Each symbol indicates one patient.
The stiffness or slackness of a muscle depends on its previous contraction level. The thixotropic response of muscle fibres results from the formation of stable crossbridges between the actin and myosin filaments, the contractile elements within a muscle fibre. If a muscle remains passive and its length is unchanged, these stable crossbridges stay intact for long periods. A consequence of the crossbridge formation is stiffening of the muscle fibres, as implied by the term thixotropy. Stretching the muscle detaches the crossbridges.

Under physiological conditions, during every blink the levator palpebrae muscle relaxes and the orbicularis oculi muscle contracts, and this causes the levator to be stretched and the crossbridges to be detached. This physiological mechanism is disturbed in patients with facial nerve palsy. Because of disturbed blinking on the side of the paresis, the levator palpebrae muscle is not stretched sufficiently and so the levator crossbridges remain intact. This will cause excessive stiffness of the levator muscle.

In a group of patients with a facial nerve palsy and lagophthalmos, we manually stretched the levator palpebrae muscle, thereby detaching the crossbridges within the muscle fibres. The effect of this stretch manoeuvre was clinically significant, as the degree of reduction in lagophthalmos achieved would be sufficient to prevent corneal ulceration.

The upper eyelid stretch manoeuvre was initially applied to investigate our hypothesis of the cause of lagophthalmos. The results appear to confirm our explanation of the pathophysiology of this condition. Additional investigations are now required to develop strategies for preventing or treating lagophthalmos. For example, it needs to be shown whether early intervention by stretching the affected eyelid could prevent the occurrence of lagophthalmos or synkinesia. Until such studies are undertaken, we are instructing our patients to close the affected eyelid passively and to stretch the levator muscle manually at repeated intervals through the day.

ACKNOWLEDGEMENT

We thank Professor M Vermeulen and Dr M Raasveld for their helpful comments during the preparation of the manuscript.

REFERENCES