Original Article

Dynamic Electrooculography Findings for Medial Rectus Myofascial Release in Esodeviation

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Abstract

Purpose: To determine which mechanisms are operative in releasing the extraocular myofascial tissue in response to extraocular myofascial release (EOMR) and to evaluate the effect of EOMR on saccadic velocity and esodeviation angle in patients with convergence spasm.

Methods: Fourteen patients with convergence spasm aged 20–35 participated in this research. The treatment included touching the medial rectus and its interrelated fascial tissue with the index finger pulp from over the eyelid for at least 300 s and applying very gentle and uniform pressure. We evaluated the saccadic velocity obtained from dynamic electrooculography (EOG) and the angle of deviation. The findings of dynamic EOG were used as a reliable quantitative method to assess eye movement function.

Results: The amount of esodeviation decreased significantly at both far 2.39Δ, 95% confidence interval (CI) (1.27–3.52) ($P = 0.002$) and near 5.57Δ, 95% CI (4.67–6.47) ($P = 0.001$) after two sessions of EOMR in a week. There was no significant difference in saccadic velocities before and after treatment.

Conclusion: In the short term, the EOMR only affects the static condition of the eye. Therefore, a significant improvement could be seen in the deviometric findings. However, the dynamic properties of the extraocular muscles did not improve and probably needed a more extended treatment period for acting the long-term mechanisms.

Keywords: Convergence spasm, Esodeviation, Extraocular muscles, Extraocular myofascial release, Saccadic velocity

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Submitted: 22-Jul-2023; **Revised:** 03-Aug-2023; **Accepted:** 03-Aug-2023; **Published:** 21-Dec-2023

Introduction

Myofascial release is a well-known and effective method to restore normal structure and function in skeletal muscles.[1,2] Tissue release can be achieved through several mechanisms, such as mechanical and neurophysiological processes, which can be triggered by manipulation. Mechanical mechanisms such as thixotropy,[3] piezoelectricity,[3] fascial adhesions,[4] and fluid flow[5] operate via direct tissue response to mechanical stimulation and need a long time to accomplish. In contrast, the neurophysiological mechanisms lead to rapid tissue release because they act through the nervous system's response to the mechanoreceptors' activity.[3,6]

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Quick Response Code:

Website: https://journals.lww.com/joco

DOI: 10.4103/joco.joco_143_23

Myofascial release has rarely been used for craniofacial muscles. However, since the extraocular muscles are striated muscles like skeletal muscles, and due to the extensive connective tissue within the orbit, it is anticipated that myofascial release will also prove to be beneficial for the extraocular muscles. Although there are similarities between extraocular and noncranial skeletal muscles, they differ in their structure and function. These include differences in muscle fiber size and arrangement, the ratio of fast to slow-tonic fibers, myosin heavy chains expression, and fatigue resistance.[7] Thus, we anticipate that the response of the extraocular muscles to myofascial release will display distinct characteristics.

How to cite this article: Mohamadi A, Vasaghi-Gharamaleki B, Mirzajani A, Jafarzadehpur E. Dynamic electrooculography findings for medial rectus myofascial release in esodeviation. J Curr Ophthalmol 2023;35:190-4.

It remains uncertain which mechanisms are effective in releasing the extraocular myofascial tissue and to what extent each works. For the first time, we developed a myofascial release technique to improve the function of the extraocular muscles; and used it to recover the esodeviation caused by convergence spasm. In addition to deviometry, we performed an electrooculographic examination before and after treatment to better understand the effective mechanisms of extraocular myofascial release (EOMR). Analyzing the saccadic velocity obtained through electrooculography (EOG) can provide valuable insights into the functioning of the extraocular muscles.[8-10]

Methods

This self-controlled clinical trial study has been approved by the Ethics Committee of the Iran University of Medical Sciences, a branch of the Iran National Committee for Ethics in Biomedical Research (IR.IUMS.REC.1399.1241), and adheres to the principles of the Declaration of Helsinki. The clinical trial is registered in the Iranian Registry of Clinical Trials, a primary registry in the World Health Organization registry network (IRCT20210130050183N1).

This study included participants with comitant esophoria/esotropia at far or near, even after receiving complete refractive correction and showing a normal gradient accommodative convergence/accommodation (AC/A) ratio (mean ± standard deviation [SD] of 2.86 ± 2.40).[11] These participants must have experienced deviation or related symptoms for <6 months. The study did not include patients who had suppression in Worth's four-dots test, any underlying disease or drug use, a history of head or eye trauma, or a history of strabismus surgery. Individuals with more than +3.00 diopters of cycloplegic hyperopia were excluded from the study, regardless of whether it was corrected or uncorrected.

Figure 1: The position of the medial rectus. When the patient abducts his right eye, the anterior portion of the medial rectus is accessible for extraocular myofascial release (a), the circle shows the cornea position, and the rectangle shows the medial rectus position beneath the closed eyelid (b)

After measuring the patients' deviation at far and near, the gradient AC/A ratio was calculated with −2.0 diopters lenses. Finally, cycloplegic refraction was done with cyclopentolate 1% eye drop. If there was ≤+0.50 uncorrected hyperopia after cycloplegic refraction, the patient could be a candidate for treatment intervention.

To perform the EOMR technique, we asked the patient to abduct one eye while the eyes were closed [Figure 1]. Then the medial rectus and its interrelated fascial tissue were touched with the index finger pulp from over the eyelid [Figure 2] for at least 300 s by applying a very gentle (a few grams) and uniform force.[1] Then, the same was done for the other eye. On the other hand, to ensure no significant pressure on the globe, the patient was asked to immediately report any feeling of compression, pain, or any colored aura caused by the pressure (phosphene sensation). If this happened, the force was reduced and kept as light as a touch. The EOMR technique was performed by a single investigator for all participants.

The medial rectus spasm could be felt at the fingertip during the technique. Likewise, in the continuation of the process, the real-time monitoring of the release of the muscle and its interrelated fascia was possible by the tactile sensation; so that the stiff tissue of the spastic muscle became soft and elastic.

After selecting the patients to enter the study, far (4 m) and near (40 cm) deviation angles were measured by the cover test and prism-bar (Luneau, Pont-de-l'Arche, France) and a target of 20/25 single optotype. Furthermore, the patient was subjected to a dynamic EOG test (MonPackOne, Metrovision, Pérenchies, France) to evaluate the horizontal saccades' velocity once when the targets were 10° and again when they were 20° apart. We recorded 15 saccadic movements for each condition to measure the saccadic velocity. Then, we averaged the peak velocity of all 15 measurements to obtain a mean saccadic peak velocity for each patient. Nevertheless, for simplicity, we used the term saccadic velocity instead of the mean saccadic peak velocity throughout the article. Two EOMR treatment sessions were accomplished with an interval of 1 week. After 5 min, the deviometry and dynamic EOG were repeated, and the results were compared with the pretreatment findings.

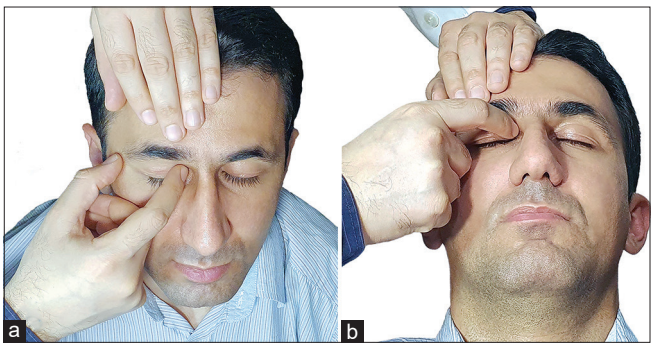

Figure 2: Extraocular myofascial release technique. High-angle (a) and low-angle (b)

Statistical analysis was performed using IBM SPSS Statistics Version 25.0 software (IBM Corp., Armonk, NY, USA). The Shapiro–Wilk test was used to test the normality of data distribution. Then, the paired-samples *t*-test or related-samples Wilcoxon Signed Ranks Test was used to compare the before and after treatment findings based on the data distribution. A statistical significance level of 0.05 was considered.

Results

Seventeen patients aged 20–35 with esophoria or esotropia resulting from convergence spasm met the criteria to be included in this study. However, three were excluded from the study because they did not adhere to the protocol (missed a therapeutic session). Therefore, 14 patients (12 women) aged 26.8 ± 5.9 years old participated in this study (mean ± SD). Their cycloplegic refraction was −0.95 ± 1.24 diopters (spherical equivalent), and their AC/A ratio was 2.57 ± 0.92 (mean ± SD).

The initial measurements of the esodeviation were 5.46Δ ± 7.72 at far and 7.82Δ ± 6.67 at near distances (mean ± SD). After the treatment, the amounts of esodeviation were 3.07Δ ± 5.93 at far and 2.25Δ ± 6.08 at near distances (mean ± SD). The amount of esodeviation decreased significantly, as much as 2.39Δ, 95% confidence interval (CI) (1.27–3.52) ($P = 0.002$) for far and 5.57Δ, 95% CI (4.67–6.47) ($P = 0.001$) for near after two sessions of EOMR in a week.

There was no significant difference in the velocity of saccades before and after treatment, neither for the right nor left eye, and with any degrees of EOG target separation [Table 1 and Figure 3].

Discussion

This study indicated that EOMR significantly improves the esodeviation angle. However, it does not have a remarkable impact on the saccadic velocity. This finding means that although the muscle's tonus and static function are improved, it is hard to pinpoint an enhancement pattern in the muscle's kinetics after two sessions of EOMR for the spastic medial recti. To find out the reason, we should investigate the factors that may influence the static and kinetic functions of the ocular motility system.

The fascia is rich in smooth muscle fibers, enabling it to contract efficiently. It has been proved that the autonomic nervous system controls the smooth muscle's contraction.[12] Stimulation of mechanoreceptors in the connective tissue decreases sympathetic nervous system activity, which leads to smooth muscle relaxation. As a result, fascial tissue can be released thanks to rapid neural mechanisms.[3,13] Given that fascia influences positional characteristics more than movement traits,[14,15] deviometric outcomes are expected to improve after EOMR.

On the other hand, the velocity of saccades is primarily influenced by the extraocular muscles rather than the fascia. This is because the passive force from the antagonist muscle's viscosity is the primary load on an extraocular muscle.[16] In addition, the proper functioning of muscles is closely related to their passive characteristics, such as length and elasticity.[1] The regeneration of collagen fibers that enhances muscle elasticity and improves muscle contracture occurs through long-term mechanisms.[17,18] Thus, spastic muscle tissue requires a more prolonged release period. Therefore, it is only logical to expect changes in saccadic velocity after long-term mechanisms have taken effect.

The dynamics of antagonist muscles differ in slow and fast eye movements. The motoneurons that innervate the antagonist muscle are entirely turned off during fast movements.[19] As a result,

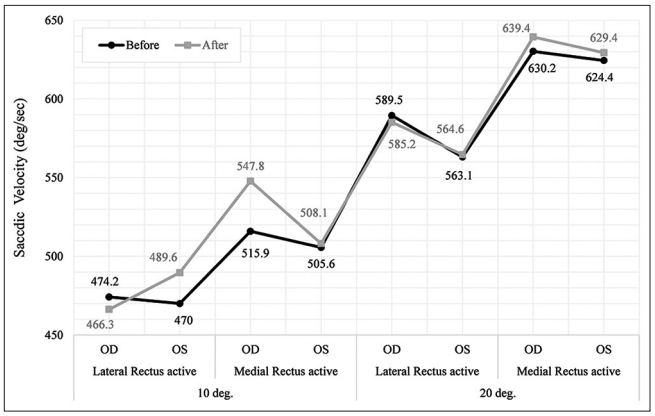

Figure 3: Mean saccadic velocities (black numbers represent before and gray numbers represent after treatment values). OD: Right eye, OS: Left eye

Table 1: Mean differences of saccadic velocity before and after treatment

EOG targets setting	Active muscle	Eye	Mean±SD	95% CI of the difference	*P*
10° apart	Lateral rectus	OD	7.89±59.7	−26.57±42.34	0.629
		OS	−19.65±79.6	−65.58±26.29	0.372
	Medial rectus	OD	−31.94±93.1	−85.71±21.83	0.222
		OS	−2.43±79.4	−48.24±43.39	0.911
20° apart	Lateral rectus	OD	4.31±55.2	−27.57±36.19	0.775
		OS	−1.56±95.1	−56.45±53.33	0.730
	Medial rectus	OD	−9.25±97.0	−65.28±46.77	0.727
		OS	−5.00±65.1	−42.60±32.60	0.778

EOG: Electrooculography, CI: Confidence interval, SD: Standard deviation, OD: Right eye, OS: Left eye

only the passive properties of the antagonist work against the muscle activity. However, both muscles' motoneurons are active during slow movements or static conditions, resulting in push-pull behavior.[19] Consequently, the early-acting neural mechanisms of myofascial release may impact the eye's static conditions more significantly than the dynamic ones. The effect of EOMR on passive muscle properties seems insufficient in the short term. Thus, the saccadic velocity would not increase significantly since the instant muscle release is due to neural mechanisms.[3,13]

Separate neural pathways can be another reason for our posttreatment findings. Erkelens *et al*. found that there are distinct neural processing circuits for dynamic and tonic vergences. Specifically, the cerebellar areas are responsible for regulating dynamic vergence, while tonic vergence remains unaffected by them.[20] This study's findings suggest that, at least in the short term, EOMR may only influence muscle tone and associated control centers while not impacting the parameters that determine the saccadic velocity.

We can also interpret the findings of this study from a cellular standpoint. Unlike skeletal muscles, extraocular muscles' myofibers do not course from tendon to tendon.[7,21-23] During the EOMR procedure, it is only possible to access the anterior portion of the medial rectus. Therefore, we anticipate that the muscle fibers originating from the other side of the muscle and terminating before reaching the globe not be released by the EOMR. This feature can also explain the unpredictability of extraocular muscle force in some laboratory conditions.[24]

Besides, due to the presence of both nerve ending types (i.e., en plaque and en grappe) in many extraocular muscles' myofibers, different regions of a single myofiber may have dissimilar contractile properties.[25] As a result, it is even thought that EOMR may only influence a part of some myofibers. Jacoby *et al*. showed that fast myosin heavy chain is expressed in the middle of the myofibers with multiple innervations, where the en plaque endplates are placed. In contrast, the slow-tonic myosin heavy chain is expressed at both ends of the fibers, where the en grappe endplates are located.[25,26] Electrophysiological studies also confirmed that the middle portions of these fibers have spiking responses and twitch-like characteristics, while tendon endings exhibit tonic characteristics instead.[25,26]

From the neural point of view, those oculomotor motoneurons receiving afferent projections from the presaccadic areas project to singly innervated twitch fibers located in the muscle's mid-belly. On the other hand, another group of nerve fibers that do not receive saccadic premotor afferent projections innervate multi-innervated nontwitch fibers at the distal ends of the muscle.[27] This finding suggests that, since the medial rectus tendon end is manipulated in EOMR, only the tonic properties of myofibers with multiple innervations are affected. The myofibers' middle portion activity is necessary for saccadic movements, the region we cannot access during EOMR.

Twenty percent of the orbital and 10% of the global layer of the extraocular muscles' myofibers are slow-tonic.[28] According to our findings, convergence spasm probably involves slow-tonic fibers more, and EOMR mainly affects these fibers. As a result, we found a significant improvement in posttreatment deviometry, where the role of slow-tonic fibers is more decisive. Nevertheless, no apparent difference was seen in saccadic velocity, where the fast fibers' role is more prominent. In addition, alterations in slow-tonic fibers, which account for a minor percentage of extraocular muscle myofibers, seem to have a negligible effect on saccadic movements, so this change is not reflected in the EOG results.

The different geometrical structures of extraocular muscles' myofibers may also account for the findings of this study. Extraocular muscles contain a considerable amount of branched fibers. This complexity creates a nonlinear resultant force in the motor units.[22] In addition, the complex serial and parallel connections between myofibers and the diversity of myosin expression in myofibers[29,30] may alter the outcome of EOMR from a simple linear pattern to a more complex one.

It should be noted that the condition known as spasm of convergence is not very common. Therefore, the number of cases included in this study was limited. Additionally, EOMR is a relatively new therapeutic method, and practitioners may need to learn the correct procedure to achieve the best results. Thus, the treatment outcome may depend on the operator to some extent. However, the most significant limitation of this study was the short duration of treatment, so it is suggested that further research is conducted over a more extended period to fully understand the long-term effects of EOMR.

In conclusion, our findings suggest that in the short term, EOMR only affects the static characteristics of the extraocular muscles. Therefore, an improvement can be seen in the deviometric results. Although improving the static conditions and alignment may raise the potential for enhancing the dynamic functions, it is necessary to continue treatment for a more extended period to improve the eye dynamics. At the same time, considering some exercises for saccades, pursuits, and vergence facilities may improve kinetic functions.

Financial support and sponsorship

This project was supported by the Iran University of Medical Sciences.

Conflicts of interest

There are no conflicts of interest.

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