

Electromyography of Human Saccadic Eye Movements

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The purpose of this paper is to report the results of an electromyographic study of saccadic movements (rapid versions) performed during a previously reported study of coactivity of the human extraocular muscles.¹ The technique employed has been previously described.²

During a saccadic eye movement in any direction, there is increased electrical activity of all muscles except the one reciprocally inhibited by the required movements. The evidence for this is as follows:

In Figure 1, the arrows from above indicate the start of horizontal saccades, the first to the left, the second to the right. During each saccade, there is increased electrical activity in both vertical recti, suggesting cocontraction. The agonist during the first saccade (right medial rectus) manifests increased activity. After the saccade the activity of each of the muscles returns to the level required by the final

position of the eye. The first muscle channel (right lateral rectus) should be disregarded for the time being. It is not working because of a poor insertion.

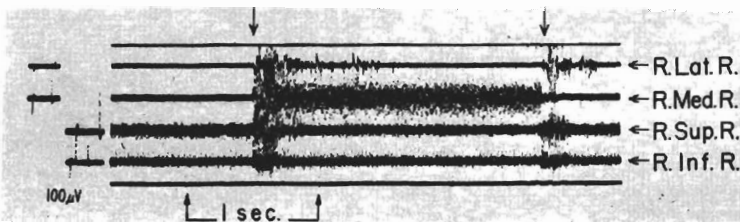
Cocontraction is defined as a simultaneous contraction of muscles which are normally antagonistic in their primary field of action. The apparent coactivity of the vertical recti and the increased activity of the agonist during the saccade, as in Figure 1, could not be immediately accepted because of the many artifacts inherent in electromyography of the extraocular muscles.³ It is necessary to differentiate actual sustained motor unit activity from possible artifactual activity. We have seen how artifacts resulting from blinking (arrows, Fig. 2) can grossly suggest short bursts of motor unit activity. But blink responses are shorter in duration than the saccadic response and, in addition, characteristically give rise to slower potentials which appear as shifts in baseline (Fig. 2). Returning to Figure 1, we suspect that the potentials in Channel 1 during the saccade are artifactual, since we know we have a very poor insertion in the lateral rectus. We have evidence of its artifactual character rather than its being unit activity in that the medial rectus is inhibited during the

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Fig. 1.—The arrows indicate start of a saccade, the first to the left, the second to the right. Channel 1 (right lateral rectus) is a poor insertion and not recording properly. Calibration is at left, and time scale is on bottom channel.



Note increased activity of agonist (right medial rectus, first saccade) and auxiliary muscles (right superior and right inferior rectus) during the saccade and artifactual activity of first channel. Time scale shown here also applies to Figures 2, 3, and 4.

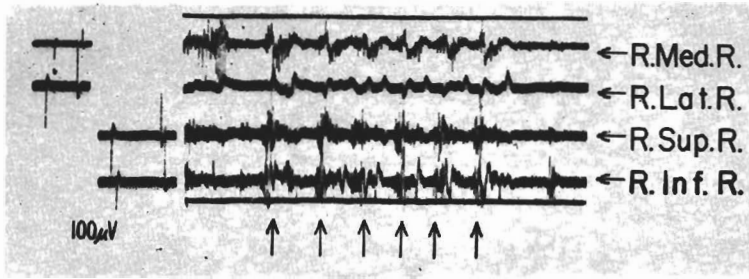


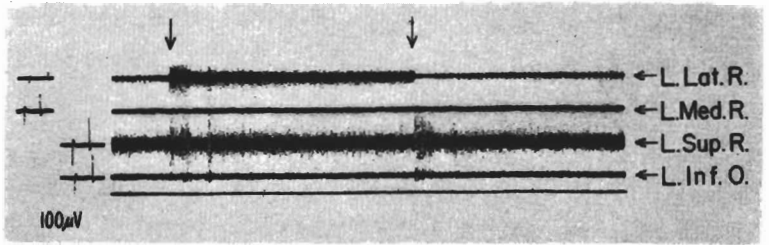
Fig. 2.—Arrows indicate eyelid blink. Note artifactual shifts in baseline and short bursts of "pseudoactivity" with each blink.

second saccade in Figure 1. Here, with a good insertion, no simulated activity occurs. If the apparent surge of activity in the agonist and auxiliary muscles were an artifact, then one would also expect the inhibited muscle (right medial rectus, second saccade, Fig. 1) to exhibit a similar increase in "activity." Further evidence is shown in Figure 3, in which there is no

lateral rectus) shows a sustained typical burst of motor activity during a saccade as compared to the irregular artifactual activity picked up by conjunctival electrodes inserted near but not into the vertical recti (Channels 3 and 4, Fig. 4).

Further elucidation of the nature of the apparent increased electrical activity in all muscles except the inhibited ones during a

Fig. 3.—Arrows indicate start of saccades, first to left; second to right. Left medial rectus has a procaine-induced paralysis. Note increased activity of agonist (left lateral rectus, first saccade) and cocontraction of auxiliary (left superior rectus) during saccades but no activity, artifactual or otherwise, from electrode in procainized muscle. Channel 4 (left inferior rectus) is a poor recording because of poor insertion.

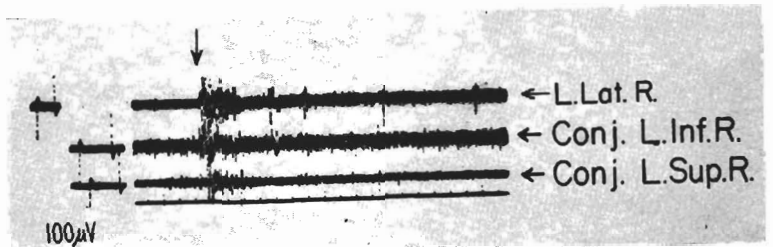


artifactual activity during saccades (arrows) in a procainized muscle (Channel 2*) or in a muscle with a poor insertion (Channel 4). Furthermore, in Figure 4 the agonist (left

saccade is obtained by running the recording film at high speed during eye movements. Artifacts, such as occur with a blink, appear mainly as a succession of large shifts in baseline on high speed recording (Fig. 5). On the other hand, the trace of the agonist (Fig. 6) or auxiliary muscles (Fig. 7) during a saccade reveals a surge of typical motor unit activity.

*The left eye actually moves to the right despite procaine-induced paralysis of the left medial rectus. A discussion of the possible reason for movement of this eye in face of paralysis of the agonist is outside the scope of this paper.

Fig. 4.—Arrow indicates start of saccade to the left. Channel 1 represents standard electrode inserted into left lateral rectus. Channels 2 and 3 represent standard electrodes inserted under conjunctiva near left inferior rectus and left superior rectus. Compare sustained activity in the agonist (left lateral rectus) with the artifactual irregular changes in Channels 2 and 3 during the saccade.



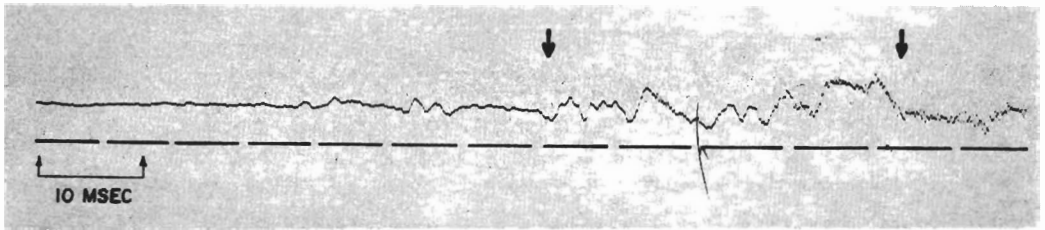


Fig. 5.—Fast film speed recording (1.5 meter per second) of a blink. It consists mainly of a series of large baseline shifts. Time scale here also applies to Figures 6, 7, and 8.

Björk⁴ also noted increased activity of an auxiliary muscle, the inferior oblique, during rapid horizontal movements of the eye.

Therefore, we can conclude that during a saccade there is a burst of activity in the agonist, complete inhibition of the antagonist, and coactivity of the auxiliary muscles.† After the saccade is

† Because of the practical anatomical difficulty in obtaining a successful insertion into the superior oblique muscle under normal conditions, we make the likely assumption that this muscle behaves as do the other auxiliary muscles.

completed, all of these muscles appear to return to the level of activity that they would normally assume for that position of the eyeball.

The duration of saccades of varying degrees of excursion can be measured by combining electro-oculography (EOG)⁵ with electromyography as in Figure 6. One need only measure the time elapsed between the start and the end of the slope of the EOG trace. This can be readily accomplished with the long and gently rising

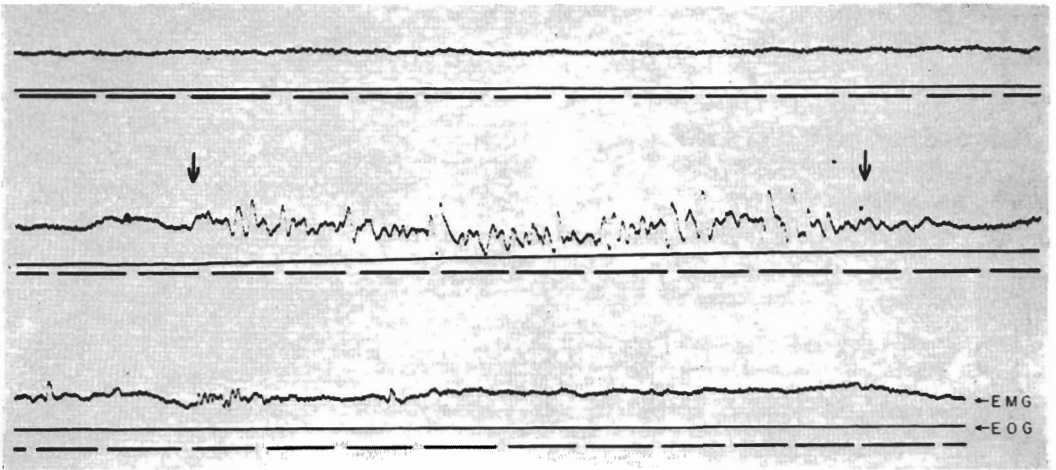
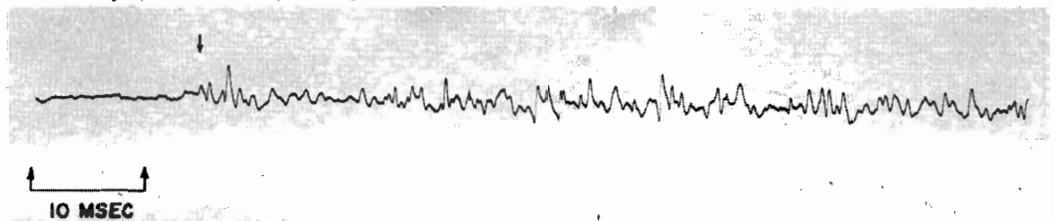


Fig. 6.—Fast film speed recording (2 meters per second) of agonist during a 26-degree saccade. Arrows indicate start and end of saccade. Lower trace is EOG. Note increased, essentially unchanging motor unit activity during saccade. The dash line is a horizontal reference line with which EOG slope can be compared.

Fig. 7.—Fast film speed recording (2 meters per second) of an auxiliary muscle (right medial rectus) during a vertical saccade. Arrow indicates start of saccade. Note increased activity (cocontraction) of right medial rectus during saccade.



TABLE

Angle of Saccade, Degrees	Mean Duration, Msec.	Mean Deviation *	N	Mean Angular Velocity, Degrees/Sec.
13	35.7	1.2	9	364.1
26	51.8	4.9	7	501.9
39	68.5	--	1	569.3
53	100.9	1.9	5	525.3

* Mean deviation equals the sum of the deviations, ignoring the sign, of each measurement from the mean, divided by the total number (N) of measurements.

EOG trace obtained with fast film-sweep speeds (2 m. per second in Fig. 6) and a D. C. amplifier. Average durations for various degrees of excursions obtained from repeated measurements in two subjects are listed in the Table.

Fast-film-speed electromyography directly confirms Westheimer's analysis which shows that saccadic eye movement is not ballistic.⁶ Braking or stopping of the movement does not come about by decreased contraction of the agonist and active increased contraction of the antagonist towards the end of the movement as seen in Figures 6 and 8, where there is no apparent decrease in activity of the agonist during the saccade (Fig. 6) and no recordable increase in activity of the antagonist during the length of the saccade (Fig. 8).

A recent paper on this same subject⁷ implies that there are two parts to a saccadic movement, an initial burst of activity

in the agonist followed by a more uniform, orderly, pattern of motor unit activity. Examination of our high-film-speed saccade tracings reveals no apparent variation in unit activity during the saccade. Comparing the durations of the initial bursts recorded in the other paper (30 to 150 msec. for saccades of 2.5 to 40 degrees) with the saccade duration reported above in the Table, it is likely that what this recent paper describes as an initial burst is the entire saccade which is then followed by the level of sustained activity for that position no matter how the eye gets there. Neither do we find evidence of a second saccade following initial saccades of more than 15 degrees, as reported.

Summary

During a saccade or rapid version movement of the eye, there is a heightened burst of activity of the agonist, inhibition of the antagonist, and coactivity of the auxiliary extraocular muscles.

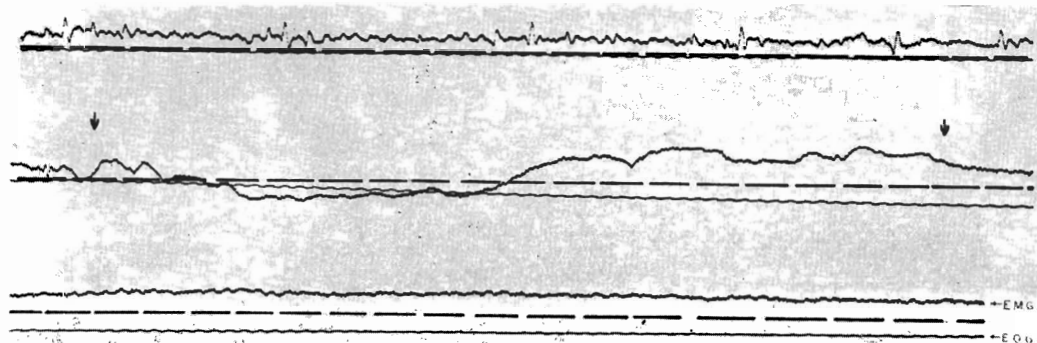
The duration of saccades of different degrees of excursions has been measured by recording simultaneously electro-oculography and electromyography.

Evidence is presented to show that saccadic eye movements are not ballistic.

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Stanford University Hospital, Clay & Webster Sts. (15).

Fig. 8.—Fast film speed recording (2 meters per second) of the inhibited antagonist during a saccade. Arrows indicate start and end of saccade. Note no significant increase in activity during saccade. The dash line is a horizontal reference line for comparison with EOG slope.



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