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Saccadic trajectories change with amplitude, not time

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ABSTRACT. Saccadic trajectories were examined in ten young normal subjects for amplitudes of 5, 15, 30, and 40 deg. Multiple saccades (m) and discrete decelerations (dd) were seen not in conjunction with any fatigue effect but rather as a function of amplitude, being much more frequent at larger amplitudes. The presence of m and dd in the large saccades of normals and in even small refixations of patients with myasthenia and Eaton-Lambert syndrome suggests that they are a normal consequence of high innervation levels.

Key words: eye movements; saccade; fatigue

INTRODUCTION

When evaluating the saccadic eye movements of both normal subjects and patients, researchers most often measure such characteristics as accuracy, peak velocity and latency. Significant information may also be found in the 'fine structure' of such movements, as well. For example, saccades may be mis-interpreted as being slow when they are actually composed of closely-spaced multiple eye movements, each of normal velocity (Bahill *et al.*, 1981). Such fragmentation of saccades has been attributed to fatigue (Bahill & Stark, 1975). These changes have been described as multiple saccades (m) and discrete decelerations (dd) (Schmidt, 1977). The former consist of several closely-spaced saccades, occurring within 70 ms; the latter are defined as discrete changes in the deceleration portion of the saccadic trajectory (Schmidt *et al.*, 1980). They have also been observed in the saccades of patients with peripheral neuromuscular disorders such as myasthenia gravis (Schmidt *et al.*, 1980) and Eaton-Lambert syndrome (Dell'Osso *et al.*, 1983). In these disorders, these trajectory changes have been attributed to both proprioceptive response to an inadequate saccade (Schmidt *et al.*, 1980) and to intermittent conduction block at the neuromuscular junction (Feldon *et al.*, 1982). It is apparent that careful analysis of the trajectory as well as the metrics of saccadic eye movements may provide significant information as to how saccades are programmed in both clinical and normal populations.

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METHODS

Data from ten subjects (two males and eight females, ages 15-32) were examined for this study. After informed consent was obtained, eye movements were recorded using infrared reflection (Narco BioSystems Model 200) and displayed on a modified Beckman Type R Dynograph with a system bandwidth of DC to 100 Hz (both position and velocity). To permit observation of multiple saccades and discrete decelerations, a chart speed of 25 mm/sec was used. This system has been previously shown to be sufficiently sensitive to detect even small multiple saccades and discrete decelerations (Schmidt *et al.*, 1980). Blinks were detected using an a.c.-coupled vertical EOG lead.

Targets were red light-emitting diodes mounted on a 1.5 meter radius arc placed the same distance from the seated subject, who was fitted with a chin rest and head brace. The test protocol was as follows: after calibration, targets were presented under computer control in the sequence left-center-right-center for 5 and 15 degrees and left-right-left-right between the 15 and 20 deg lights; thus, saccades at 5, 15, 30, and 40 deg were elicited, with all stimuli being within the ± 20 deg linear range of the recording system. For each of the target amplitudes 20 cycles were presented, with a two-minute rest being allowed between amplitudes. The 5 and 15 deg targets remained on for 1.8 sec; the 30 and 40 deg for 2-4 sec. The two smaller amplitudes were chosen to fall within the range which has been claimed for the majority of saccades made in normal life (Bahill *et al.*, 1975), while the larger two were selected to lie outside this range, thus entailing more effort. Targets were presented in order of increasing amplitude, with one subject rerun in the opposite sequence. If a blink occurred during a saccade, that target was repeated. The recordings were monitored during the session and if gross abnormalities were noted (*e.g.*, failure to respond to a target or slow, irregular movements suggestive of falling asleep), verbal encouragement to follow the target was given.

The records were examined for the presence of multiple saccades and discrete decelerations. When multiple saccades were seen, it was noted whether they occurred during the rising or falling phase of the overall saccade velocity profile. Data for both eyes of all subjects were pooled, with the exception of the run made in reverse order with one of them.

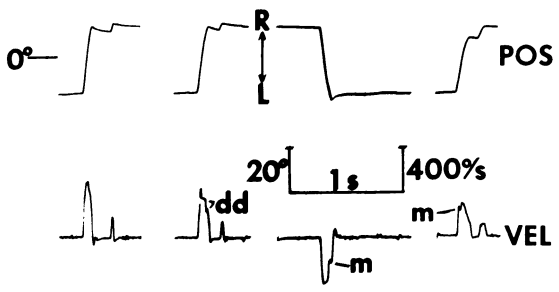

Fig. 1. Examples (going left to right) of saccades with no fragmentation, with discrete decelerations (dd) and with multiple saccades (m) occurring on the falling and rising phases of the velocity profile. POS = position, VEL = velocity, R = right, L = left.

RESULTS

As has generally been the case when testing normal individuals, a wide range of responses was observed (Boghen *et al.*, 1974; Schmidt *et al.*, 1979, 1980). A clear trend was observed, however, for the decomposition of saccades into closely spaced submovements, *e.g.*, multiple saccades and discrete decelerations (Fig. 1), to be a function of *amplitude* and not of time. This can be seen in Fig. 2, where the number of such movements seen during each 5-saccade interval can be seen for the entire subject population. Nearly all

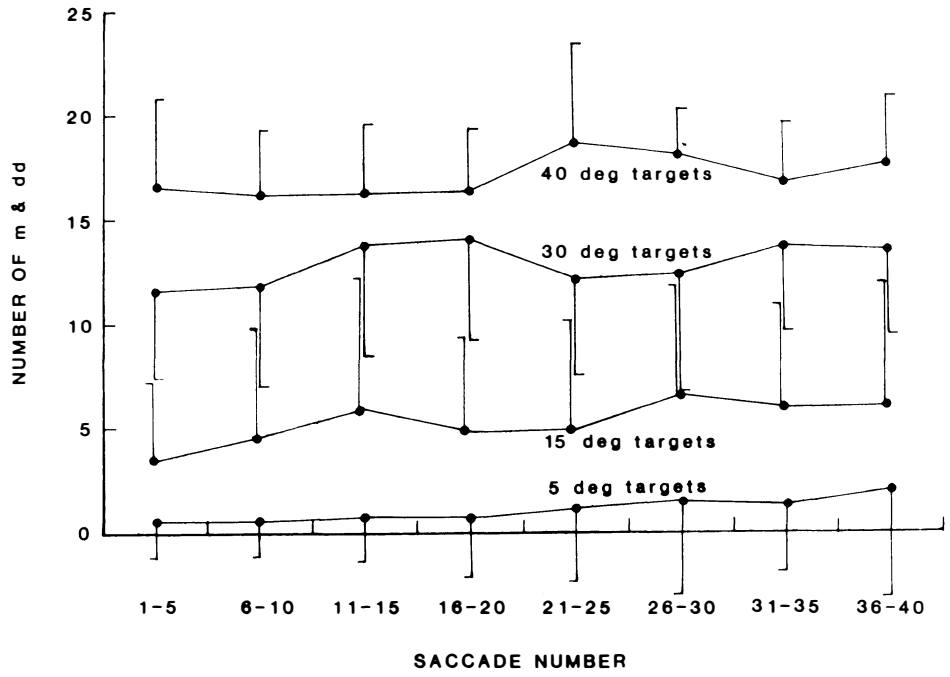

Fig. 2. Plot of number of m and dd for each target amplitude versus saccade number (thus, versus time). Data from all subjects are pooled. Standard deviations are given for each point. The number of m and dd depended much more strongly on saccade amplitude than on time.

the m and dd were binocular events, although an m in one eye was sometimes matched by a dd in the other. The number of m and dd increased only slightly during the course of a given target sequence; in contrast, there was a clear increase with increasing amplitude. Most of these complex velocity profiles were discrete decelerations or multiple saccades occurring when the overall movement was already decelerating. Only infrequently did the second saccade occur when the first was still accelerating. No relationship was observed between baseline noise levels and presence of m and dd. The subject with the highest number of them had one of the most noise-free records. Further, if m and dd were due to ongoing noise, their number should have increased in proportion to saccade duration; instead, for an approximate doubling of duration as amplitude went from 5 to 30 deg, the number of m and dd increased eight-fold. The one subject most prone to falling asleep during testing was rerun with the stimuli in reverse order. Her incidence of m and dd remained most closely tied to target amplitude, not time. Since the completion of this study, we have observed identical m and dd in recordings made using the scleral search coil technique. This rules out the possibility that they were generated by some non-linear characteristic of the infrared system.

DISCUSSION

This study assessed how saccadic trajectories changed with both time and amplitude. No gross

alterations in other saccadic measures such as velocity or accuracy over time were expected, since Fuchs & Binder (1983) found no fatigue effect on these even under circumstances which greatly stressed the ocular motor system (*e.g.*, 31 min of 60 deg saccades). Their use of bitemporal EOG for recording precluded evaluation of the details of saccadic trajectories, however. Since trajectory changes have been reported as a result of fatigue (Bahill & Stark, 1975; Schmidt *et al.*, 1979), have been suggested as a putative cause of saccadic variability (Bahill *et al.*, 1981) and have been observed in several neuromuscular disorders (Schmidt *et al.*, 1980; Feldon *et al.*, 1982) we felt it important to study the fine structure of saccades in normal subjects over a wide range of amplitudes. Pronounced fatigue effects have been reported to occur with as little as 1 min of repetitive saccade generation (Bahill *et al.*, 1981); thus, while the present study is not an exhaustive examination of long-term saccadic changes, it did examine trajectories in detail over several minutes of saccade execution.

We found no evidence that the number of m and dd increased rapidly with time, even for 40 deg saccades. The data for the one subject run in reverse order showed the same trends as those taken in order of ascending amplitude; this argues against the increase in m and dd being due to some cumulative fatigue building over the course of the session. Instead, saccadic trajectories changed with increasing amplitude. An extra acceleration (m) frequently occurred when large (30 or 40 deg) saccades were made repeatedly. These were seen on both the rising and falling phases of the velocity profile, but more often on the latter. In the deceleration phase of a saccade, presumably when the primary burst is declining, discrete decelerations (dd) frequently occurred in a similar fashion (Schmidt *et al.*, 1980). These appear as small discontinuities in the velocity tracing. The m and dd appear to form a continuum; one saccade may show a plateau in the velocity trace (dd), while the next may exhibit a small velocity increase (m) at a similar point in its trajectory. Thus, occurrences of both are summed in Fig. 2. The occurrence of m and dd mostly near the end of the saccade meant that most of the refixation was carried out by the first component. Thus, computer-generated peak velocities would still be generally accurate (that is, two closely-spaced 15 deg saccades of normal velocity would not be mistaken for one slow 30 deg saccade), so that the spurious low velocities and saccadic variability postulated by Bahill *et al.* (1981) would not be a problem. This is significant for those laboratories using computers for the analysis of eye movements, since most saccade detection and measurement algorithms would ignore the presence of m and dd.

Multiple saccades (Bahill & Stark, 1975) and glissades (Bahill & Stark, 1975; Schmidt *et al.*, 1979) have been reported to become more frequent with fatigue; however, we found that the incidence of m and dd depended instead on amplitude. As pointed out by Fuchs & Binder (1983) this fatigue resistance may well be related to the large safety factor provided by the 'overdesign' of the ocular motor system, since extraocular muscle has to lose 90% of its force before saccadic velocity is affected (Magoon & Scott, 1982). Thus, under normal conditions even large saccades would not be expected to show pronounced changes over time.

A link with pathophysiology lies in the circumstances under which high innervation levels are observed. The m and dd saccades are seen in large amplitude refixations in normals and the saccades of patients with ocular myasthenia (Schmidt *et al.*, 1980) and Eaton-Lambert syndrome (ELS) (Dell'Osso *et al.*, 1982). These conditions produce saccades of at least normal veloc-

ity, often with complex trajectories. While it had previously been suggested that the fragmentation of myasthenic and ELS saccades involved extraocular muscle proprioception (Schmidt *et al.,* 1980; Dell'Osso *et al.,* 1983), a more parsimonious and unifying explanation, based on the results of the present study, would be that the neural activity generated for a saccade normally becomes more asynchronous at high levels of innervation. Since both large saccades of normals and small saccades of patients with myasthenia and ELS require high firing rates in large numbers of motoneurons, the presence of fragmented trajectories in these instances may indicate that the firing patterns of individual agonist motoneurons are somewhat desynchronized. Since the m were generally seen in the deceleration phase, and the dd are defined as occurring there, this desynchronization may be most evident during the periods of maximal firing rate needed to sustain large saccades. If neural activity were less erratic during lower innervation levels, m and dd would be expected to be infrequent during the initial portions of large saccades and throughout smaller ones, whose firing rates never reach maximal levels. Thus, high levels of ocular motor innervation *per se* are likely to produce m and dd, regardless of their origin. This variability also extends to the innervation of each muscle, thus accounting for the occasional monocular m and dd seen. Frequent binocular, simultaneous m and dd would seem more likely to have a central, rather than peripheral, origin. The presence of m and dd in large saccades of normals and small saccades of myasthenia and ELS patients also is consistent with a central origin for them in the patient groups, rather than the peripheral conduction block proposed by Feldon *et al.* (1982). Since it seems unlikely that neuromuscular transmission becomes intermittent for large saccades in normal subjects, only the desynchronization of high innervation levels provides a consistent explanation for m and dd in all populations.

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