Saccade initiation and the reliability of motor signals involved in the generation of saccadic eye movements

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Abstract

We examined the trial-by-trial relationship between the metrics of saccadic eye movements and the activity of individual putative premotor neurons in the paramedian pontine reticular formation (PPRF) of rhesus monkeys. The region of the pons containing these excitatory burst neurons (EBNs) extends for several millimeters. Motoneurons innervating extraocular muscles integrate the output of hundreds or even thousands of these neurons. Accordingly, there was no reason to expect that relatively small variations in the activity of a single pontine neuron would be related to variations in saccade amplitude or speed observed during repetitive eye movements to the same target. Nonetheless, we observed consistent relationships between variations in the number of spikes in the burst of pontine neurons and the amplitude of the saccade. Trial-to-trial variations in the instantaneous spike frequency during a burst are associated with variations in the velocity profile of the movement. Based on these data, we conclude that the activity of pontine burst neurons is not statistically independent and that simultaneous recordings from multiple cells will reveal a high degree of correlated activity.

Keywords:

saccade
superior colliculus
paramedian pontine reticular formation
excitatory burst neurons
movement initiation
decision to move
signal reliability
signal variability
correlated signals
independent signals

I. Saccade initiation

The neural mechanisms involved in the execution of a saccadic eye movement are fairly well understood (Sparks 2002). A saccade occurs when the motoneurons innervating the extraocular muscles produce a sudden, but brief, increase in discharge rate. This “pulse” of motoneuron activity produces the transient increase in muscle tension needed to move the eye at saccadic velocity. The size and speed of saccades are controlled by the amplitude and duration of the pulse. A step change in the firing rate of motoneurons produces the steady state muscle tension needed to hold the eye at the new location in the orbit.

The signals producing the pulse and step of activation of motoneurons are constructed by feedback circuits found in the brainstem (Moschovakis et al 1996, Scudder et al 2002). Excitatory burst neurons (EBNs) located in the pons and rostral midbrain produce the pulse of activation. An instantaneous frequency plot of the high frequency burst is shown in Fig. 1A (top left). The axons of EBNs project directly to the motoneurons innervating extraocular muscle fibers. The number of spikes in the burst of activity is highly correlated with the amplitude of the movement. Also, various temporal properties of the burst are correlated with temporal aspects of the saccade: burst onset is tightly coupled to saccade onset; burst duration is highly correlated with saccade duration; and the peak velocity of the saccade is related to the peak frequency of the burst (Fig. 1A, right).

The activity of EBNs is controlled, in turn, by omnipause neurons (OPNs). OPNs are clustered together (Buttner-Ennever et al 1988) in a small midline nucleus (nucleus raphe interpositus). They discharge at a relatively constant rate during fixation intervals but stop firing in association with saccades in all directions (Fig. 1A, bottom left). The cessation of activity (pause) begins before saccade onset, before the onset of the EBN burst, and ends before the saccade is terminated. OPNs produce monosynaptic inhibition of EBNs (Nakao et al 1980, Curthoys et al 1984, Strassman et al 1987) and saccades can be interrupted in mid-flight by microstimulation of the OPNs (Keller 1974, 1977, King & Fuchs 1977).

The question of saccade initiation now becomes: “what is the ‘trigger’ signal that turns off the OPN cells?”. The superior colliculus (SC) is a major source of inputs to the pulse-step generator circuits. Neurons in this midbrain structure form a map of saccadic eye movements that can be revealed using microstimulation methods. Electrical stimulation of the intermediate and deeper layers of SC of monkeys produces conjugate, contralateral saccades with amplitudes and directions that depend on the site of collicular stimulation (Robinson 1972). The site of stimulation within the map determines the largest movement that can be produced. However, for any stimulation site as the duration of the stimulation train increases, movement amplitude increases monotonically until it reaches the site-specific limit. Additionally, the peak velocity of the evoked movement is influenced by the frequency of stimulation; higher frequencies produce movements with higher velocities. The effects of train duration and frequency can be varied to produce movements that have comparable amplitudes but different velocity profiles. Similarly, stimulation parameters can be adjusted to evoke movements of the same amplitude with different latencies. These data (Stanford et al 1996) indicate that at

least three independent signals are derived from the spatial and temporal pattern of collicular activity - one specifying the desired displacement, another related to saccadic velocity, and a third involved in the initiation of a saccade.

One particular type of neuron found in the SC, the saccade-related burst neuron (SRBN), is thought to play a key role in saccade initiation. These neurons may display a low frequency prelude of activity before a saccade, but 18-20 msec before saccade onset, the low-frequency activity is replaced by a high-frequency burst of activity (Fig. 1C). In behavioral situations in which saccadic reaction time varies over large ranges, the onset of the burst of SRBNs is tightly coupled to saccade onset (Fig. 1C). Moreover, the occurrence or lack of occurrence of the high-frequency burst can be used to predict perfectly the behavior of a monkey performing a behavioral task in which the probability of saccade initiation is manipulated by varying target duration (Sparks 1978). The axons of SRBNs form a major efferent pathway from the SC to subsequent oculomotor premotor neurons (Raybourn & Keller 1977, Moschovakis et al 1996). These and other findings form the basis of the hypothesis (Sparks 1978, Keller 1979, Sparks et al 2000) that the burst produced by SRBNs is the signal that triggers the initiation of a saccade. The high frequency burst produces a momentary release from the tonic inhibition of pontine OPNs and allows EBNS to generate a pulse of activity that is transmitted to the motoneurons. Exactly which intervening neurons are involved in converting the bursts of SRBNs into an inhibition of OPNs is unknown. Viable hypotheses exist (see, e.g. Strassman et al 1986, Scudder et al 1988) but are difficult to test experimentally.

Much contemporary research is focussed on the important question of how (from a vast array of potential targets) a particular target is selected as the goal for a saccadic eye movement. Current evidence suggests that information about the location of the selected target gradually develops during the reaction time interval (Sparks et al 1987) and that saccades are initiated when the time integral of cortical or collicular activity exceeds a threshold value (see Schall 2003, 2004 for reviews). The high frequency burst of collicular saccade-related burst neurons provides a precise indication of when the threshold is exceeded. But the mechanism by which activity accumulating in cortical and subcortical areas is suddenly translated into the burst of SRBNs to trigger a saccade has not been specified.

II. Reliability of motor signals involved in the generation of saccadic eye movements

The responses of visual neurons have been shown to be highly reproducible when the same time-varying luminance patterns are presented repeatedly (e.g., Reinagel & Reid 2000). Few comparable studies of the reliability of the motor command signals carried by individual neurons exist. In sensory studies, the physical properties of the stimulus can be held constant from trial to trial. How do we perform the motor equivalent of repeatedly presenting the same physical stimulus? If we ask a subject to make many saccades from the same initial fixation target to the same eccentric target, considerable variability in the amplitude, duration and velocity of the movement is observed (Fig. 2A). Variability in initial fixation position (center row, left) is smaller than variability in the position reached after the primary saccade (bottom row, left). Thus, small changes in the retinal eccentricity of the target stimulus resulting from variations in initial fixation position

cannot fully account for the variability in final, postsaccadic position. What are the neural sources of variability in saccade amplitude?

Methods. We examined the trial-by-trial relationship between the amplitude of saccadic eye movements and the activity of putative[1] EBNs in the paramedian pontine reticular formation (PPRF) of rhesus monkeys. These cells were chosen for study because of the strong relationships between cell activity and the parameters of the movements illustrated in Fig. 1A. Action potentials were recorded from individual pontine burst neurons while monkeys made horizontal saccades to 2 to 5 visual targets, always starting from the same initial eye position. Initially, spikes were recorded as time stamps with 1 microsecond resolution using an electronic window discriminator to determine the occurrence of an action potential. The size of action potentials becomes smaller and irregular in amplitude as the burst of pontine cells progresses (Keller 1974). Thus, the window discriminator may fail to detect one or more of the smaller action potentials in the burst. Spuriously long interspike intervals would be recorded and expressed as a "dropout" or momentary lower frequency in the instantaneous frequency records we used for the analyses presented in Figures 3-5. To avoid this potential error in measurement, we have begun recording the waveform of action potentials and checking the validity of the window discriminator output offline. These recordings have convinced us that many of the "dropouts" are artifactual. Accordingly, the data presented in this paper are based on trials in which bursts with two or more dropouts are excluded.

Number of spikes and saccade amplitude. Figure 2B plots the number of spikes generated by a single pontine neuron during 115 saccades to one target located 20 deg from the fixation point and 115 saccades to another target located 30 deg from the fixation point. Because of the variation in both the amplitude and number of spikes, the data appear as two local clusters. Within each cluster, saccade amplitude varies over a 2 to 3 degree range and the number of spikes varies by 8 to 10 spikes. The correlation coefficients between number of spikes and saccade amplitude in the two clusters were 0.50 and 0.43. The distribution of correlation coefficients obtained from 36 experiments similar to the one illustrated in panel B is presented in panel C. The average correlation coefficient for these 36 data sets was 0.37.

Some of the variability in saccade amplitude observed when the animal makes repeated movements to a particular target is associated with variability in the number of spikes produced by pontine burst neurons. Under the conditions of this experiment, we assume that all pontine EBNs are receiving a common input specifying the same desired horizontal movement amplitude. We also assume that the actual amplitude of the executed movement is based on a summation of the output of all the cells. The correlation between an EBNs number of spikes and saccade amplitude tells us how much of the variance in saccade amplitude is explained by the activity of the neuron. The variance explained is the square of the correlation coefficient. The mean correlation coefficient of our sample is 0.37; the average proportion of variance accounted for by one neuron is about 14%. Using the rationale employed by sensory neurophysiologists

1. For the cells we recorded, the relationship between measures of spike activity and saccade amplitude, duration and velocity meet criteria for classifying them as EBNs. But, by definition, EBNs project monosynaptically to motoneurons and we do not know if the cells we recorded do so. For this reason, we call them putative EBNs.

when trying to estimate how many cortical cells are involved in a perceptual decision, about 7 statistically independent pontine neurons like those recorded would account for the variance in saccade amplitude. However, we know that the excitability of the motoneuron pool is influenced by hundreds or thousands of pontine burst cells. We conclude, therefore, that the activity of pontine neurons is not statistically independent and that simultaneous recordings from multiple cells will reveal a high degree of correlated activity.

Variability in the profile of the burst produced by putative EBNs. Number of spikes was used as an index of trial-to-trial variability in the activity of pontine burst cells for the analysis presented in Fig. 2. Number of spikes is a single global measure of a time varying process. An analysis that examines variability during the burst is presented in Figure 3. Multiple traces of eye position (P), eye velocity (V), and instantaneous spike frequency (F) are superimposed in panels A-E. The mean and standard deviation of instantaneous frequency was calculated for 2 msec bins for the bursts associated with 10, 20, and 30 degree horizontal movements. The standard deviation of instantaneous frequency is plotted in the bottom trace of panels A-E. The plots in panels A-C are aligned on movement onset and plots in panels D and E are aligned on burst onset.

Note that the largest variability in burst activity is associated with the onset and end of the burst. During the sustained part of the burst, the standard deviation of instantaneous frequency is often less than 10% of the firing rate. The average standard deviation for the shaded regions of the plot in panel A was 48.7 spikes/sec, about 6% of the average frequency (785 spikes/sec) during this period (coefficient of variation = 0.06). Standard deviations and percent of average frequency for the shaded regions in panels B and C were 63.5 spikes/sec (9%) and 63.2 spikes/sec (9%). The variability occurring at burst onset is significantly reduced if trials are aligned on spike onset (compare A vs. D and B vs. E).

The average velocity profiles and average instantaneous frequencies for the 10, 20 and 30 deg movements shown in Panels A-C are superimposed in Panel F. The average velocity profiles follow a common trajectory for different distances, depending on saccade amplitude. For 20 and 30 deg saccades, this cell's firing rate changed almost instantaneously to the same peak frequency and then declined gradually until near the end of the burst when a sharper reduction in frequency occurs. The instantaneous frequency profiles are similar for most of the duration of the bursts associated with 20 and 30 deg movements. The variability (standard deviation) during the middle segments of the bursts associated with 20 and 30 deg movements may be comparable (panels B,C) because the cell is firing at about the same frequency during these intervals.

The data presented in Figure 4 further examine sources of variability in the burst profile of pontine burst cells. Position, velocity, instantaneous frequency and standard deviation of instantaneous frequency are plotted in panel A for 22 saccades to a target located 10 deg from the fixation stimulus. The plots on the left were aligned on movement onset and those on the right were aligned on burst onset. Aligning the plots on burst onset significantly reduces variability of the initial segment of frequency records, but there may be a concomitant increase in variability near the end of the burst. An algorithm for sorting movements by similarity of the velocity profile was used to identify the two subsets of the movements illustrated in panels B and C. For these panels, the standard deviation plots is a measure of variability around the mean frequency profile

computed for each subset of movement, not variability around the mean for all 22 movements. One consequence of selecting a subset of movements with similar velocity profiles is a reduction in the variability of saccade duration and associated reductions in the variability of instantaneous frequency near the end of the burst. The standard deviation of instantaneous frequency during the middle segment of the burst was 8-9% of the frequency for the data presented in panel A. This was reduced to 5-6% for the subsets of movements shown in panels B and C.

The data presented in Fig. 4D are consistent with the suggestion that much of the variability in the burst of pontine neurons for saccades of a given amplitude and direction occurs during the increase in firing rate at the beginning of the burst and the decrease in discharge rate near the end of the burst. Velocity and instantaneous frequency plots are superimposed for 27 movements. The area under the standard deviation plot for the twelve 30 deg movements is shaded to facilitate comparison with the plot of the standard deviation of the instantaneous frequency for the fifteen 40 deg saccades. The variability in instantaneous frequency is similar during the middle, sustained portion of the bursts. Also, the increase in variability occurring near the end of the bursts is larger for the 40 deg movements. This increase in variability may correspond to the larger period over which individual burst frequencies decay to zero and this, in turn, may determine a larger range in the time when the velocity of individual movements returns to zero.

These preliminary findings lead to the following speculations. As illustrated in Figure 5A, when a subject makes repeated saccades from the same fixation target to the same eccentric target, considerable variability is observed in final position and velocity profile. The burst of putative EBNs activating the appropriate motoneuron pools also displays variability, as illustrated by the plot of average instantaneous frequency and the surrounding curves representing one standard deviation boundaries. However, if subsets of movements with distinguishably different velocity profiles are selected, the instantaneous frequency plots are also distinguishably different. The movements highlighted by darker lines in the top trace of Fig. 5B reach higher peak velocities and have shorter durations than the subset of movements highlighted in the plot in the second row. The peak average instantaneous frequency is greater and burst duration is shorter for the plots associated with the subset of movements having higher peak velocity and shorter duration. Such a relationship between the activity of a single pontine burst cell and the velocity profile of a subset of movements would not be observed if other members of the large active population of EBNs had quite different temporal profiles of burst activity or if, in general, the burst profiles were heterogeneous and uncorrelated. Thus, this preliminary analysis of the variability in the burst profile of pontine neurons supports the conclusion reached based on the correlation between number of spikes and saccade amplitude - variability in the discharge of a single EBN is not independent, but strongly correlated with the activity of other active EBNs.

Further research is needed to: determine the validity of eliminating “dropouts” from the data analysis; verify the preliminary results presented in this paper suggesting a deterministic relationship between the precise temporal patterns of firing of an EBN and the particular velocity profile of a saccade; and, ascertain to what extent the correlation between the activity of different pontine cells originates from common inputs and/or from synaptic connections between EBNs.

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Figure Legends

Figure 1. Role of pontine and collicular neurons in saccade initiation. A. left. Instantaneous frequency plots of the saccade-related activity of an EBN (top) and OPN (bottom). P: horizontal position. V: horizontal velocity. right: plots of the relationships of measures of the activity of an individual EBN with saccade latency, amplitude, duration and velocity. B. left, plots of horizontal eye position for 9 saccades ranked in order of saccade latency. center, raster plots of the activity of a SRBN during the 9 movements. right, relationship between burst latency and saccade latency.

Figure 2. Behavioral and neuronal variability during repeated saccades to the same visual target. A. Left. plots of horizontal position (top), initial fixation position (middle) and postsaccadic position (bottom) for 57 saccades to a single target. For each, the monkey was instructed to produce a saccade 18 degrees in amplitude by looking from an initial fixation target 10 deg to the right of the straight ahead position to an eccentric target appearing 8 deg to the left. Right. Observed variability in horizontal velocity (top), duration (middle) and peak velocity (bottom). B. Correlation between number of spikes and saccade amplitude observed during repeated movements to two different saccade

targets (see text for additional detail). The long line represents a line of best fit between number of spikes and saccade amplitude for saccades to different targets. The shorter lines represent lines of best fit obtained by a linear regression of saccade amplitude onto burst size saccade amplitude for each cluster of saccades to the same target. C. Distribution of correlation coefficients obtained from 36 measurements similar to those illustrated in panel B.

Figure 3. Variability in the temporal profile of EBN bursts. Plots of horizontal position (P), velocity (V), instantaneous spike frequency (F), and the standard deviation of instantaneous frequency during thirteen saccades 10 deg in amplitude (A), twelve 30 deg movements (B), and fourteen 20 deg saccades (C). Instantaneous frequency is the reciprocal of interspike interval, measured by counting the number of 1 microsecond clock pulses that occurred between adjacent action potentials. The plots in panels A, B, and C were aligned on saccade onset. Plots aligned on the instantaneous frequency plots are shown in panels D (10 deg movements) and E (30 deg movements). Note the reduction in the initial segments of the plots of standard deviation when trials are aligned on burst onset (A vs. D; B vs E). The average values of the standard deviations for the shaded areas in plots A,B and C are given in the text. F. Plots of the average velocity and instantaneous frequencies for the 10, 20, and 30 deg movements plotted in panels A-C.

Figure 4. Factors contributing to the variability in the burst profile. A. Superimposed plots of horizontal position (H), velocity (HVel), instantaneous spike frequency (Freq), and the standard deviation of the instantaneous spike frequency (SD) for 22 saccades approximately 10 deg in amplitude. Plots are aligned on saccade onset (left) or on burst onset (right). Note the reduction in the variability of instantaneous frequency during the initial segment of the burst when trials are aligned on burst onset. B. Superimposed plots of velocity, frequency and standard deviations for a subset of 11 trials selected on the basis of the similarity of the velocity profiles. C. Same as B but for a different subset of 6 trials. D. Superimposed plots of horizontal velocity (first row) and instantaneous frequency (second row) for multiple saccades to visual targets located 5, 30 and 40 deg from the fixation stimulus. Standard deviations of the instantaneous frequency of the bursts preceding the 30 deg (shaded curve) and 40 deg (solid line) movements. See text for more details.

Figure 5. Comparison of instantaneous spike frequencies for movements of the same amplitude but differing in velocity profile. A. Superimposed plots of horizontal position (H) and velocity (HVel) for 22 saccades to the same target (20 deg eccentricity) are plotted above the average instantaneous frequency and curves showing +/- 1 standard deviation of two msec segments of the curve. B. Two subsets of the 22 movements having different velocity profiles are highlighted in the top two plots. The bottom row shows the average instantaneous frequencies associated with each subset of movements. See text for additional details.

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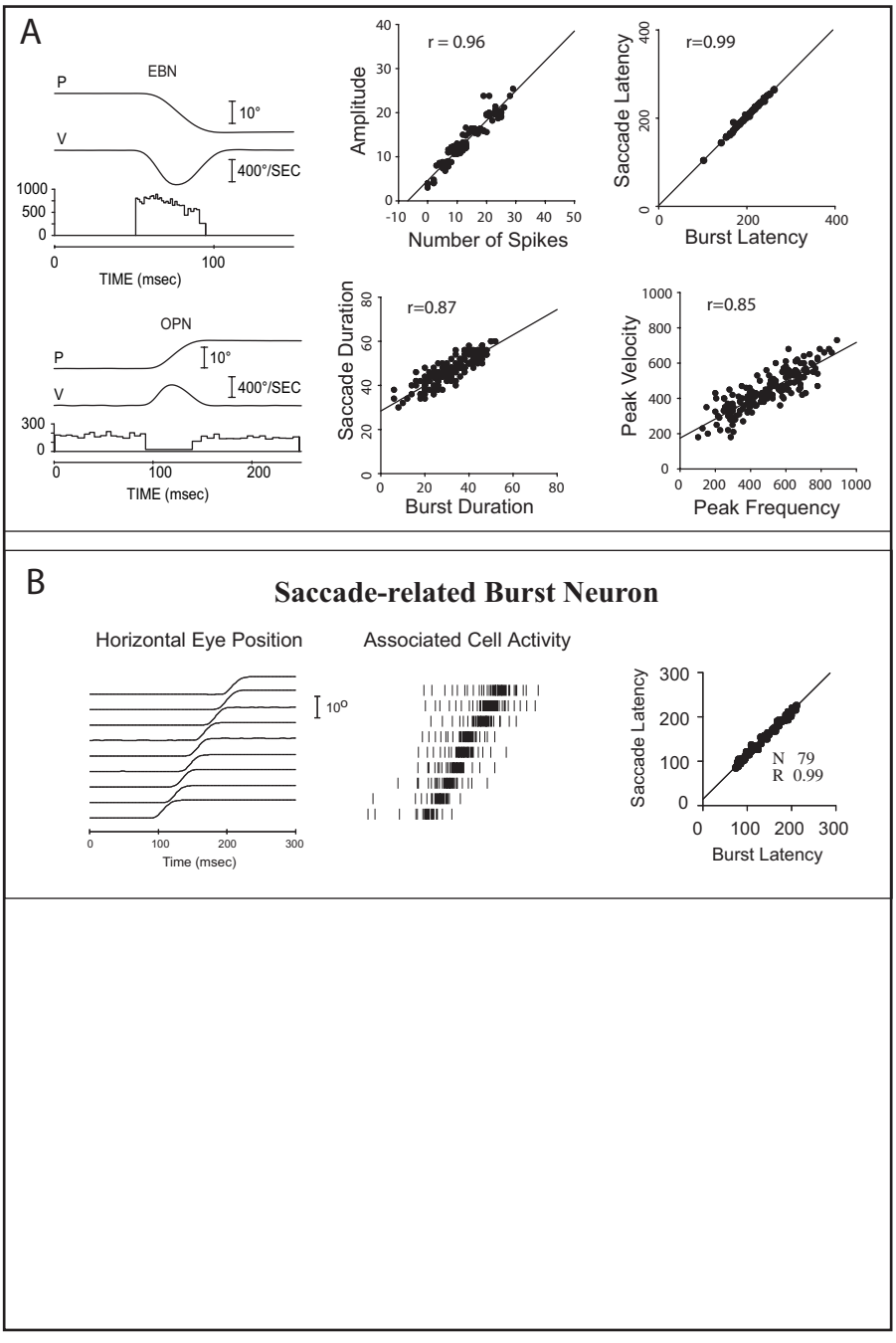
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EBN
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100
TIME (msec)
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P
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V
400°/SEC
300
0
0
100
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TIME (msec)
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Amplitude
Number of Spikes
r=0.99
Saccade Latency
Burst Latency
r=0.87
Saccade Duration
Burst Duration
r=0.85
Peak Velocity
Peak Frequency
B
Saccade-related Burst Neuron
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Associated Cell Activity
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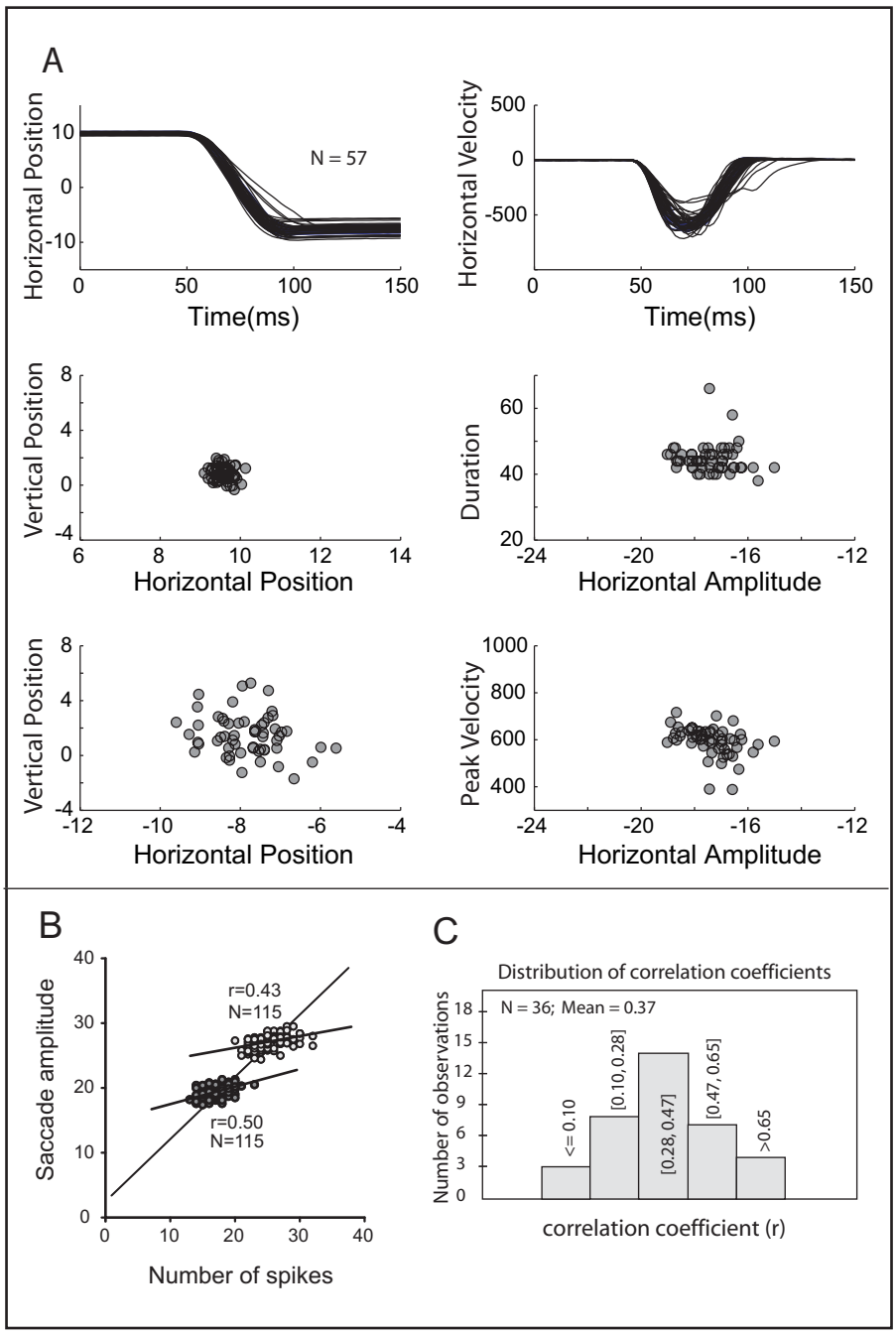
Burst Latency

Fig 1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Horizontal Velocity
Time(ms)
Vertical Position
Horizontal Position
Duration
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Vertical Position
Horizontal Position
Peak Velocity
Horizontal Amplitude
B
Saccade amplitude
Number of spikes
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N=115
r=0.50
N=115
C
Distribution of correlation coefficients
N = 36; Mean = 0.37
Number of observations
correlation coefficient (r)
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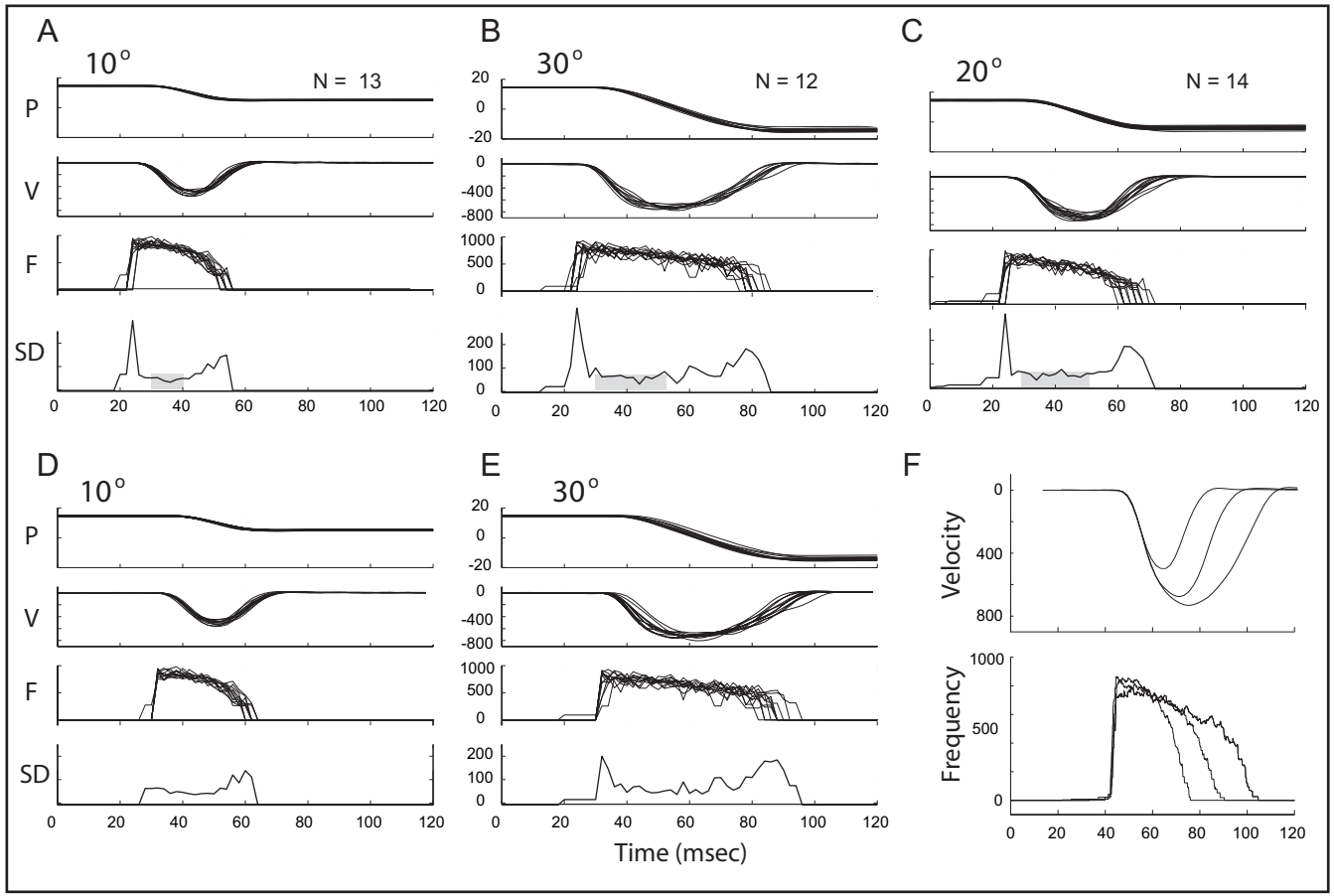
[0.28, 0.47]
[0.47, 0.65]
>0.65

Fig. 2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P
V
F
SD
B
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C
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D
10°
E
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F
Velocity
Frequency
Time (msec)

Fig. 3

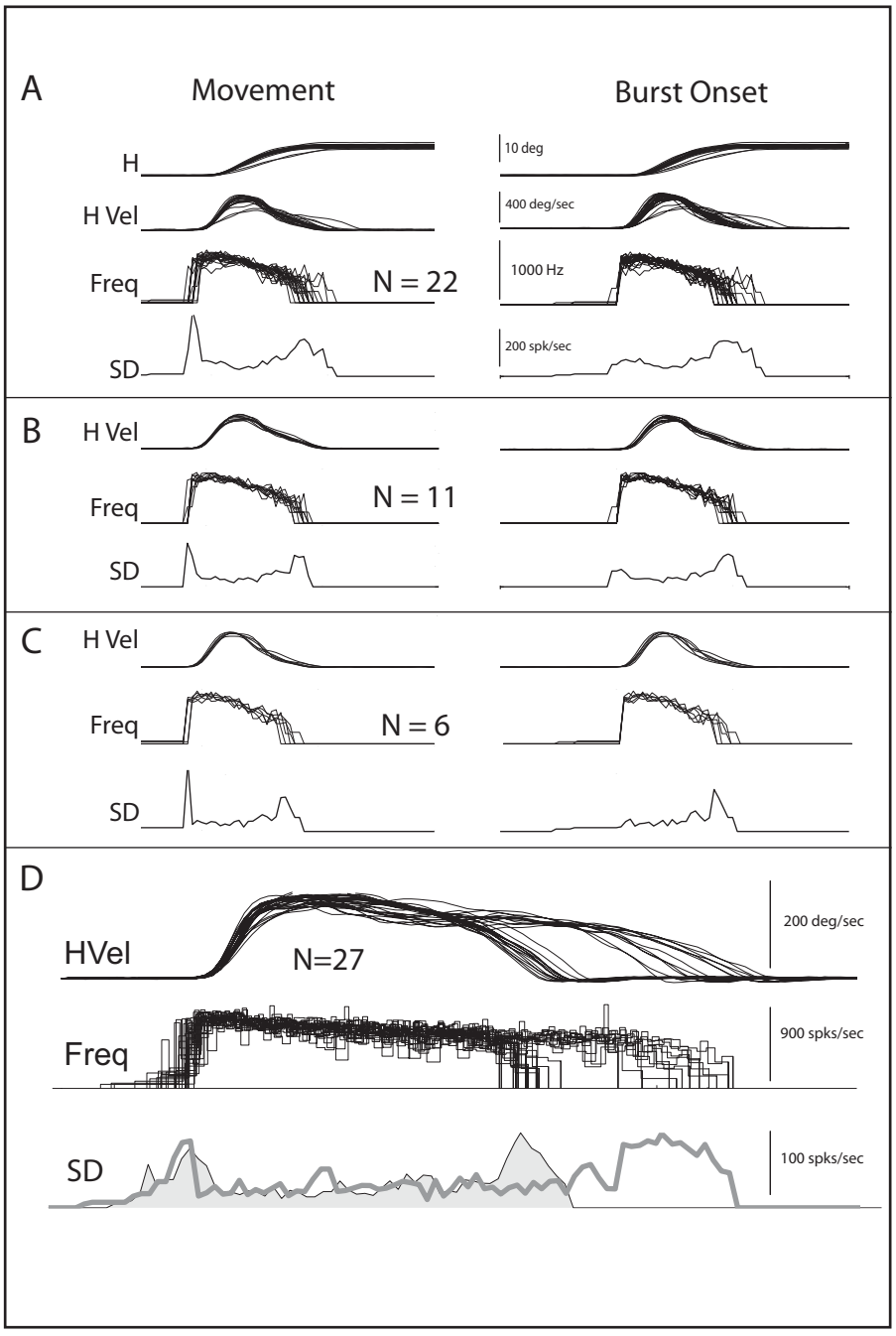
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Fig. 4

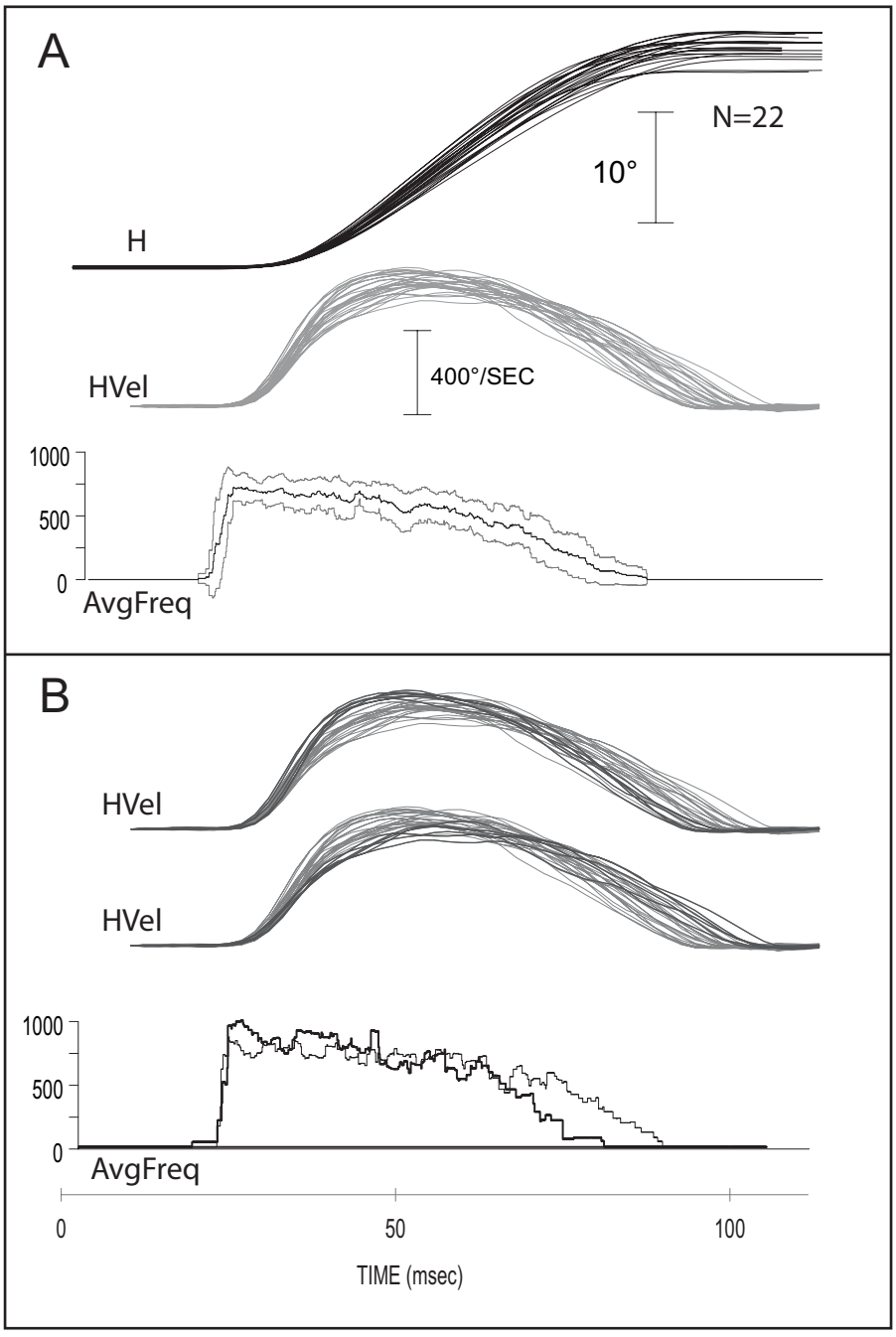

Fig. 5
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