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7 Synchronizing the tracking eye movements with the  
8 motion of a visual target: Basic neural processes

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27

28 **Abstract**

29 In primates, the appearance of an object moving in the peripheral visual field elicits an  
30 interceptive saccade that brings the target image onto the foveae. This foveation is then  
31 maintained more or less efficiently by slow pursuit eye movements and subsequent catch-up  
32 saccades. Sometimes, the tracking is such that the gaze direction looks spatiotemporally  
33 locked onto the moving object. Such a spatial synchronism is quite spectacular when one  
34 considers that the target-related signals are transmitted to the motor neurons through multiple  
35 parallel channels connecting separate neural populations with different conduction speeds and  
36 delays. Because of the delays between the changes of retinal activity and the changes of  
37 extraocular muscle tension, the maintenance of the target image onto the fovea cannot be  
38 driven by the current retinal signals as they correspond to past positions of the target. Yet, the  
39 spatiotemporal coincidence observed during pursuit suggests that the oculomotor system is  
40 driven by a command estimating continuously the current location of the target, i.e., where it is  
41 here and now. This inference is also supported by experimental perturbation studies: when the  
42 trajectory of an interceptive saccade is experimentally perturbed, a correction saccade is  
43 produced in flight or after a short delay, and brings the gaze next to the location where  
44 unperturbed saccades would have landed at about the same time, in the absence of visual  
45 feedback. In this chapter, we explain how such correction can be supported by previous visual  
46 signals without assuming “predictive” signals encoding future target locations. We also  
47 describe the basic neural processes which gradually yield the synchronization of eye  
48 movements with the target motion. When the process fails, the gaze is driven by signals related  
49 to past locations of the target, not by estimates to its upcoming locations, and a catch-up is  
50 made to reinitiate the synchronization.

51 **Keywords**

52 Tracking, saccade, pursuit, foveation, neurophysiology, synchronization, prediction

53

54 Over the course of their phylogenetic and ontogenetic evolution, the animals were  
55 faced with the need to manage an environment which is not composed of static objects. In  
56 their world (*Umwelt*), the significant events occur most often when something moves (whether  
57 it is animal or not, predator, prey, congeners, or other) or when themselves, they move. In front  
58 of this dynamism, the animals exhibit the ability to pursue and catch a prey, to avoid a predator,  
59 in flight for some of them. How are such performances, spatiotemporally accurate, possible?  
60 Should we believe that the functioning of the brain sets general frames (such as space and  
61 time) and principles similar to those used by the Newtonian mechanics for describing the  
62 motion of a rigid body? In this chapter, we will explain how the neurophysiology of eye  
63 movements made toward a moving visual target addresses these fundamental questions. A  
64 new line of investigation is proposed to identify the neurophysiological processes which  
65 underlie the ability to produce a visuomotor behavior which is accurate both spatially and  
66 temporally.

## 67 **1. SPATIOTEMPORAL “DILUTION” OF VISUAL SIGNALS IN THE BRAIN.**

68 Most visually guided actions consist of localizing and aiming at selected objects in the  
69 external world while avoiding obstacles, and grasping them once their location becomes within  
70 reach. They involve a multitude of saccades whereby the gaze is oriented toward selected  
71 parts of the external world. By bringing the object within the central visual field, each saccade  
72 leads to a situation where the target excites a larger population of neurons. In the visual areas  
73 of the cerebral cortex and in the superior colliculus (SC), more neurons are indeed sensitive  
74 to stimuli located within the central visual field (McIlwain, 1976). If the target is static, the  
75 primary saccade is often completed by a small secondary saccade (also called correction  
76 saccade). If the target is moving, the interceptive saccade is followed by a slow movement of  
77 the eyes and later by a small catch-up saccade. We will avoid considering this sequence of  
78 eye movements as the signature of a purposive process whereby a desired future state would  
79 drive the evolution of the current state. Instead, we will try to view it as an inevitable process  
80 unfolding in the inhomogeneous structure of the visuomotor system, through parallel streams  
81 of activity constrained by the brain connectivity.

82 From the excitation of ganglion cells in the retina to the recruitment of motor neurons  
83 (MNs) in the reticular formation and the contraction of extraocular muscles, multiple trains of  
84 action potentials are conveyed by parallel channels connecting, with different conduction  
85 speeds, assemblies of diverse types of neurons characterized by different response times  
86 (Nowak and Bullier, 1997; Pellionisz and Llinas, 1982; Schmolesky et al., 1998). The  
87 generation of saccadic and slow eye movements indeed involves streams of activity which, on

88 their way from the optic nerve toward the oculomotor nerves, mobilize several populations of  
89 neurons in the thalamus, the cerebral cortex, the basal ganglia, the cerebellum, and the  
90 reticular formation (Büttner-Ennever, 2005; Krauzlis, 2004; Lynch, 2009; Moschovakis et al.,  
91 1996; Wurtz and Goldberg, 1989). Thus, the activity yielded by the projection of a small static  
92 spot of light on the retinal surface, spatially delimited, spreads out and becomes largely  
93 distributed (spatially and temporally) in the brain networks before converging onto the groups  
94 of premotor and MNs, and causing the contraction of two pairs of six extraocular muscles  
95 (Goffart, 2017).

96 **< Figure 1 approximately here >**

97 Beyond the apparent entropy exhibited by this spatiotemporal “dilution” of afferent  
98 signals within the cerebral medium, the massive interconnectedness of neural populations  
99 actually facilitates the persistence of visual activity for a duration which largely exceeds the  
100 actual duration of even a brief spot of light in the external world. Unlike the artificial light which  
101 turns off immediately after an electric switch is pressed, the visual activity persists and  
102 continues its diffusion within the brain networks. Fig. 1 shows examples of saccades aimed at  
103 a target LED that was flashed for a short duration (50 ms) in the peripheral visual field of two  
104 macaque monkeys (Be and E). From the onset of the target to the beginning of the saccade  
105 toward its location, the saccade latency is the time interval for localized changes in retinal  
106 activity to be transmitted to the muscle fibers and initiate their contraction. The contraction lasts  
107 for a duration which is sufficient to rotate the eyes and accurately direct the foveae toward the  
108 target with a time course which does not slow down with longer latencies. Regardless of its  
109 onset time, the saccade indeed consists of a sudden increase in angular velocity (acceleration)  
110 followed by a decline (deceleration). The persistence of the target-related activity is nicely  
111 appreciated when the intensity of visual signals is reduced. Indeed, the use of LEDs allows  
112 testing the influence of target energy (Barnes, 1995), and discovering that low-intensity targets  
113 lead to saccades which are not characterized by a slower rise in eye velocity and a longer  
114 deceleration; it is their onset which is actually delayed (the latency increases). The variability  
115 of latencies also increases when the target contrast is reduced (see gray traces in Fig. 1).  
116 Interestingly, the latency of the first spike of the response of neurons in the primary visual  
117 cortex is also variable and can be correlated with the saccade latency (Lee et al., 2010). Of  
118 course, this attractive influence of such a dim spot of light has been amplified by the fact that  
119 the monkey learned that it was a signal whose foveation led to a reward. This artificial  
120 association would have hardly been observed in the wilds; such a transient stimulus would  
121 have likely been unnoticed. Beyond the rigorous description of a behavioral response, the point  
122 that we want to address here concerns the identification of the propagation of signals within

123 the cerebral medium (see also Van Rullen et al., 2005) for that specific case when an orienting  
124 response is being elaborated and made toward a visual target.

## 125 **2. SYNCHRONOUS POPULATION ACTIVITY AGAINST FUNCTIONAL** 126 **ENTROPY**

127         Considering that the action potentials are conducted along the axons of cells with fixed  
128 speeds of propagation, the difference of saccade latencies illustrated in Fig. 1 can be explained  
129 by changes in the excitability of neurons. One possible mechanism is that the number of  
130 ganglion cells, which, in the retina, are simultaneously excited, is larger with a high-contrast  
131 stimulus than with a low-contrast one. This synchronous activation would then facilitate the  
132 recruitment of postsynaptic neurons in the thalamus or in the SC, then in the visual cortex, etc.  
133 The same principle could be involved in reducing the delay of propagating signals between the  
134 relays interposed between the retina and the MNs: simultaneous excitatory signals from an  
135 assembly of neurons are more efficient in exciting their targeted cells than temporally scattered  
136 input. Thus, the variability of saccade onsets would depend upon the synchronies of action  
137 potentials which are transmitted to the next relay: weaker synchrony yielding scattered and  
138 longer reaction times.

139         Providing a direct support to this hypothesis is difficult with the contemporary  
140 electrophysiological recording techniques. However, the analysis of the effects of electrically  
141 exciting the same neural site in the SC of the macaque monkey is very instructive for that  
142 particular issue. The SC is a structure anatomically situated between the visual cortical areas  
143 of the cerebral cortex and the premotor regions in the pontomedullary reticular formation  
144 responsible for the generation of saccades (Hall and Moschovakis, 2004). In its deeper layers,  
145 one finds cells which fire selectively prior to saccades within a limited range of directions and  
146 amplitudes (Schiller and Koerner, 1971; Sparks et al., 1976; Wurtz and Goldberg, 1972). The  
147 size and the extent of this movement field depend upon the location of the cell in the SC. In  
148 the anterior region, the neurons emit a burst of action potentials during small saccades,  
149 whereas neurons located more posteriorly fire during saccades of larger amplitude. Neurons  
150 located in the medial border burst prior to upward oblique saccades, and those located on the  
151 lateral border discharge before downward oblique saccades. Interestingly, when an electrical  
152 microstimulation is applied to a particular site, a saccade is elicited with an amplitude and  
153 direction which belong to the movement field of recorded neurons (Schiller and Stryker, 1972).  
154 While the amplitude and direction of evoked saccades depend upon the stimulated site  
155 (Robinson, 1972), their latency depends upon the rate of stimulation pulses: the lower the rate,  
156 the longer the latency (Katnani and Gandhi, 2012; Stanford et al., 1996). Fig. 2A illustrates this

157 effect. For frequencies ranging from 300 to 500 pulses per second (pps), the electrically evoked  
158 saccades have identical velocity profiles (Fig. 2B) and thus, identical amplitude and direction  
159 (identical amplitudes of their horizontal and vertical components). However, their latencies  
160 increase when the stimulation frequency diminishes. When the frequency is reduced to 200  
161 pps, the latency becomes more variable (Fig. 2C). The “synchronous recruitment” hypothesis  
162 described at the beginning of this section can account for this phenomenon: increasing the  
163 frequency of stimulation favors the synchrony of firing between the electrically excited neural  
164 elements (cells and axons), and this facilitates the recruitment of targeted premotor cells,  
165 shortening the latency of elicited saccades.

166 **< Figure 2 approximately here >**

167 To our knowledge, Schiller (1970) was the first to consider the influence of the number  
168 of neurons discharging in synchrony as an important determinant of saccade generation. And  
169 this intuition leads us to another important point, i.e., that the rate of firing is not the sole  
170 parameter by which the SC activity contributes to the generation of saccades; the number of  
171 active neurons also matters. Indeed, when a subset of the active population of neurons is  
172 inactivated by the local injection of a small volume of pharmacological agent, the velocity of  
173 saccades is reduced (Lee et al., 1988) and their latency increased (Sparks et al., 1990) in a  
174 correlated manner (Goffart et al., 2012). This influence of the number of active neurons can  
175 likewise be demonstrated with the microstimulation technique by varying the current of  
176 stimulation. When the frequency and duration of stimulation are maintained fixed, increasing  
177 the current of stimulation reduces the latency of saccades (Fig. 2D; Katnani and Gandhi, 2012;  
178 Stryker and Schiller, 1975). However, contrary to a transient visual target which leads to a  
179 complete saccade long after its disappearance (Fig. 1), electrical microstimulation in the deep  
180 SC evokes a saccade whose amplitude depends upon the stimulation duration. If the  
181 stimulation is not sufficiently long, the evoked movement is truncated; the amplitude specified  
182 by the locus of activity in the SC cannot be fully accomplished (Freedman et al., 1996; Stanford  
183 et al., 1996). Sustained activity is an important aspect of the collicular involvement in  
184 generating saccades toward the location encoded by the population of active neurons. The  
185 maintenance of firing only concerns the activity generated during saccades and not the  
186 complete activity which elapses from the target onset until the saccade end. Indeed, before  
187 the emission of their saccade-related burst, numerous neurons exhibit a reduction or even a  
188 pause of their firing after their visual response (Bell et al., 2006; Mays and Sparks, 1980). The  
189 activity during saccades steers the gaze toward a location which is specified by the current  
190 population of active neurons in the SC, whether the saccade is made toward static (Gandhi

191 and Keller, 1999; Glimcher and Sparks, 1993) or moving targets (Fleuriet and Goffart, 2012;  
192 Goffart et al., 2017a; see below).

### 193 **3. REDUCING THE LAG BETWEEN THE PAST AND THE PRESENT**

194 The conduction delays between the firing of retinal ganglion cells and the burst  
195 discharge of ocular MNs make impossible that the gaze is driven by the contemporary retinal  
196 signals during its saccadic shifts. The problem becomes sharper when one considers the  
197 generation of saccades toward a moving target. If the saccades were aimed at the locations  
198 traveled by the target, they would always fall short and miss it with an error which would depend  
199 upon the target speed and the time of saccade “landing.” Yet, despite that the retinal signals  
200 yielded by a moving target always correspond to its past locations, the interceptive saccades  
201 can be as accurate, though not as precise, as saccades toward a static target (e.g., Barmack,  
202 1970; Cassanello et al., 2008; Fuchs, 1967; Keller and Johnsen, 1990; Robinson, 1965). Fig.  
203 3 compares the landing errors between saccades made toward a static target (red symbols)  
204 and saccades made toward a target moving with a constant speed of 21 degrees (deg)/s (blue)  
205 in the same region of the visual field (Fleuriet et al., 2011). After a step of 16 deg along the  
206 vertical (Fig. 3A) or horizontal meridian (Fig. 3B), the target moved horizontally or vertically  
207 (respectively), i.e., along the axis orthogonal to the step. In comparison to saccades toward  
208 the static targets, the interceptive saccades can be accurate (overlap of error values), with a  
209 tendency for more frequent undershoots (negative values of error). However, the quasi-  
210 absence of overshoots (positive values) whose size is larger than the errors of saccades  
211 toward a static target indicates that the interceptive saccades are not aimed at a future location  
212 of the target, contradicting the claim that “in [their] programming, target motion is used to  
213 predict the future target position so as to assure a spatial lead of the gaze at the saccade end,  
214 instead of attempting a precise capture of the target” (Klam et al., 2001). In fact, the idea that  
215 the brain would predict the future location of a moving target, and, accordingly, that it would  
216 issue a command corresponding to a kind of foresight (Berthoz, 2012; Optican and Pretegianni,  
217 2017) lacks of any empirical evidence.

218 **< Figure 3 approximately here >**

219 Electrophysiological recording studies teach us that any saccade made toward a static  
220 target is commanded by a population of neurons in the SC (Anderson et al., 1998; McIlwain,  
221 1976, 1991; Sparks et al., 1976, 1990). Some cells discharge maximally before and during the  
222 movement, while others fire less. Those which burst the most vigorously are located at the  
223 center of the active population, surrounded by the others. Let’s consider five hypothetical



224 neurons, A, B, C, D, and E; the neuron B firing maximally during a 10 deg rightward saccade,  
225 the neuron A during a 8 deg rightward saccade, and C during a 12 deg rightward one. The  
226 neurons D and E prefer slightly oblique rightward saccades: D when the 10 deg rightward  
227 saccade has a 2 deg upward component, and E when it has a 2 deg downward component.  
228 The activity of each neuron can be symbolized by a vector with two coordinates, each  
229 corresponding respectively to the horizontal and vertical component of the saccade during  
230 which it fires maximally: (8,0) for A, (10,0) for B, (12,0) for C, (10,2) for D, and (10, 2) for E.  
231 According to the vector-averaging hypothesis (Pitts and McCulloch, 1947; Sparks et al., 1976,  
232 1997), the direction and amplitude of the ensuing saccade is specified by the average activity  
233 of the population of firing neurons. A 10 deg rightward saccade would involve our five neurons  
234 A, B, C, D, and E. The discharge of neuron B would be the most vigorous, while A, C, D, and  
235 E would fire less. Situated outside the active population, the other neurons (e.g., F: (10,6), G:  
236 (10,-6), H: (10,4), and I: (10,-4)) would be silent. Let's consider now that an identical 10 deg  
237 rightward saccade is made toward a target which moves downward, along a path parallel to  
238 the vertical meridian. If the same vector-averaging principle steers the interceptive saccade,  
239 then a larger population of active neurons should be observed in the SC. The moving target  
240 indeed traveled locations in the upper visual field which correspond to vectors like (10,6) and  
241 (10,4), involving the activity of neurons F and H. Thus, for generating exactly the same  
242 horizontal saccade, the neurons (G and I) coding for the vectors (10,-6) and (10,-4) should also  
243 fire, even though the target did not yet "reach" their response field. In other words, these  
244 neurons should fire action potentials during interceptive saccades and not during identical  
245 saccades made toward a static target. Their movement field should exhibit a shift of their  
246 boundary in the direction opposite to the target motion direction. But the available evidence  
247 argues against this scenario; the boundaries of the neurons' response field are not shifted in  
248 the direction opposite to the direction of the target motion. Many neurons do not exhibit any  
249 shift. Other neurons do, but the shift is in the same direction as the direction of the target  
250 motion. In other words, the active population in the SC is dynamical and consists of a  
251 continuum of neurons issuing commands ranging from vectors related to the past location of  
252 the target to vectors related to its present location (Goffart et al., 2016, 2017a; Keller et al.,  
253 1996), amending the suggestion that the locus of activity in the SC would only encode the  
254 location that the target occupied in the past, at the time of its appearance (Optican, 2009;  
255 Optican and Pretegianni, 2017). Finally, the empirical data do not refute the vector-averaging  
256 hypothesis because different weights can be attributed to the spikes that the saccade-related  
257 neurons emit during the interceptive saccade.

#### 258 4. CAPTURING A MOVING TARGET HERE AND NOW

259 The majority of behavioral studies show that in response to a moving target, the gaze  
260 does not land on a location where it will be in the future. The world of living animals contains  
261 too many uncertainties that they do not behave as if their prey, predator, or congeners were  
262 moving along predetermined routes comparable to public transportation lines. The most  
263 spectacular ability with which animals are endowed is to physically capture one to several  
264 moving objects. By reducing both the delay between the detection and the reaction to a moving  
265 object, and by maintaining it within the central visual field, the object becomes quasi-stable,  
266 both spatially and temporally. Instead of considering processes which “predict” or “foresee” the  
267 future location of moving target, we propose that tracking a moving target consists of  
268 establishing spatial synchrony with it (see also Becker and Fuchs, 1985). The fact that  
269 engineers found procedures and built machines which can point toward the future location of  
270 an object does not necessarily imply that the same “computation” happens in the brain when  
271 a moving target is being captured.

272 < Figure 4 approximately here >

273 This fact that the saccades do not target the future but the current location of a moving  
274 object is strongly suggested by the results of experiments which tested the robustness of  
275 interceptive saccades in the monkey (Fleuriet and Goffart, 2012). When a saccade toward a  
276 moving target is perturbed by a brief microstimulation in the SC, the electrically induced change  
277 in eye position is corrected in flight or after a short delay, bringing the direction of gaze toward  
278 the location where unperturbed saccades would have landed at about the same time. In  
279 particular, when the microstimulation is applied in a site which is not involved in the generation  
280 of saccades with amplitude and direction close to those of interceptive saccades (when the  
281 direction of the perturbation is opposite to the saccade direction), after the electrically induced  
282 change in gaze direction, most correction saccades do not overshoot along the motion path.  
283 They either fall short or “land” accurately on the moving target. Fig. 4 shows examples of such  
284 corrections observed in one experiment (see Fleuriet and Goffart, 2012 for other examples).  
285 The experiment was composed of trials which started with the fixation of a central target for a  
286 variable interval (the latter is a methodological precaution for avoiding the generation of  
287 premature saccades). At the end of the fixation interval, the central target was turned off and,  
288 at the same time, a peripheral target appeared 12 deg along the vertical meridian (downward  
289 or upward) and moved horizontally to the left or to the right with a constant velocity of 20 deg/s.  
290 To avoid the use of visual signals in the guidance of corrections during the perturbed trials, the  
291 target became invisible 150 ms after the onset of its motion and for an interval lasting 300 ms.  
292 After this interval of target invisibility, the target reappeared moving in the same direction and

293 with the same velocity for 500 ms. The monkey's task was to make a saccade toward the target  
294 and to pursue it until it reached its destination (trial end). In 50% of the trials, the interceptive  
295 saccade was perturbed by an electrical microstimulation in the deep SC at the onset of the  
296 blank interval (i.e., 150 ms after the target motion onset). For both unperturbed and perturbed  
297 trials, a fruit juice reward was delivered to the monkey at the end of the trial. Interestingly, the  
298 microstimulation evoked saccades whose amplitude and direction were influenced by the  
299 target motion (compare the eye displacements indicated by the black arrows). When the target  
300 moved in the upper right quadrant (Fig. 4B), the stimulation evoked a saccade with small  
301 upward and rightward components. When the target moved in the upper left quadrant (Fig.  
302 4A), the horizontal component almost disappeared. When it moved in the lower left quadrant  
303 (Fig. 4C), both horizontal and vertical components were strongly reduced; the interceptive  
304 saccade was delayed. These different perturbation vectors illustrate interactions between the  
305 electrically-evoked and target-elicited activities. Their study should be informative because  
306 they illustrate the dynamics (rise and decline) of target-related signals, as previously  
307 documented with a static target (Nichols and Sparks, 1995, 1996; Schlag et al., 1998; Sparks  
308 and Mays, 1983; Sparks et al., 1987). Following the perturbation, a correction saccade was  
309 made that brought the gaze direction, not where the target will reappear (future location), but  
310 toward a location close to those captured by unperturbed saccades.

311 Two groups of signals can participate in the elaboration of the command which guides  
312 the interceptive saccade toward the transiently invisible target regardless of whether its  
313 trajectory is perturbed or not: (1) the target motion-related signals that precede the blank  
314 interval and (2) mnemonic signals that the target is expected to reappear and continue to move  
315 along the same path. As explained in the first section, the massive interconnectedness of  
316 neural populations in the brain facilitates the persistence of visual activity for duration which  
317 largely exceeds the actual duration of an event in the external world. Thus, it is quite possible  
318 that after the moving target disappears, the visual activity persists and continues its diffusion  
319 within the brain networks. Presumably, the diffusion is not isotropic but biased by the target  
320 motion direction. It has indeed been shown that the pursuit eye movement persists beyond the  
321 time and location where a moving target has disappeared (e.g., Mitrani and Dimitrov, 1978).  
322 The activity driving this persistent slow movement of the eyes is likely not restricted to the  
323 ocular MNs; it must be driven by premotor neurons, which themselves could be driven by  
324 persisting activity of visual neurons. Thus, the correction saccades observed after the  
325 "spatiotemporal" perturbation induced by electrical microstimulation in the dSC (Fig. 4; Fleuriet  
326 and Goffart, 2012) could well be driven by residual activity from the visual signals which  
327 preceded the target disappearance. Later experiments testing saccades toward a transient  
328 moving target showed indeed a large number of landing positions situated beyond the location

329 where the target disappeared. When the target moved at constant speed, the saccades landed  
330 approximately where the target would be if it were still moving during the next 100 ms (see Fig.  
331 8B, E, and H in Quinet and Goffart, 2015a). Therefore, the visual signals that persisted after  
332 the target disappearance could contribute to guide the correction saccades reported in Fleuriet  
333 and Goffart (2012) because the added delays did not exceed 100 ms (see our Fig. 4 and Figs.  
334 6 and 7 in Fleuriet and Goffart, 2012). In addition, the target disappeared for a short interval  
335 and reappeared 150 or 300 ms later, continuing its motion along the same path with the same  
336 velocity. There was no uncertainty about whether the target would reappear and keep moving  
337 along the same path. The monkeys never experienced cases where the target would start  
338 moving backward or change its direction during the interval of invisibility.

339         Moreover, the monkeys were not tested to only make a saccade toward the transient  
340 moving target (like in the study of Quinet and Goffart, 2015a). They were used to continue their  
341 tracking after the target reappearance, until the end of the trial. In other words, it is possible  
342 that additional central factors contributed to the robustness of interceptive saccades. Fig. 4  
343 indeed shows that the monkey made catch-up saccades when the target was not visible, during  
344 unperturbed trials (green arrows) but also during perturbed trials (blue arrows). Following the  
345 target disappearance, the diffusion of residual signals could merge with prelude signals related  
346 to the upcoming target reappearance. Thus, the interval of invisibility would be “filled” by a kind  
347 of interpolation, driving the activity of premotor neurons and guiding the eye movement  
348 (saccadic or slow) in the absence of target-related retinal signals. Therefore, the command  
349 which encodes at best the expected and current (here-and-now) location of the target and  
350 drives the premotor neurons when a visual target is being temporarily occluded corresponds  
351 to a combination of signals related to the recent past with mnemonic signals carrying an  
352 expectancy built upon the past and repeated experience.

## 353 **5. BEYOND THE NOTION OF INTERNAL MODEL**

354         Several studies show cases where the eyes rotate smoothly and in spatial synchrony  
355 with the motion of a visual object, as if no delay separated the time-varying changes of target  
356 location and the time-varying changes of extraocular muscle tension. The gaze direction  
357 seems to be locked onto the present location of the target, i.e., where it is here and now.  
358 Obviously it is not the retinal signals yielded by the external target which drive this  
359 spatiotemporal coincidence.

360         According to the most popular view, the ability to perform visually guided eye  
361 movements which are spatiotemporally accurate rests upon a putative internal model of the

362 moving target. The major concern of this explanation is that it leads to a dead end insofar as it  
363 refers to an entity which is neurophysiologically intractable. Nowadays, the notion of internal  
364 model is used like a shortcut, barely helpful to identify and characterize the underlying  
365 neurophysiology. The major obstacle which compromises this possibility is the distributed  
366 nature of the representation of a visual target in the brain activity (e.g., Nowak and Bullier,  
367 1997; Schmolesky et al., 1998; Sparks et al., 1976). A moving visual target involves flows of  
368 neural activity which do not remain local but propagate both “vertically” (i.e., across the  
369 successive relays interposed between the retina and the extraocular MNs) and “horizontally”  
370 (diffusion within each relay). In other words, in the brain activity, the neural image (in the  
371 mathematical sense) which corresponds to an object in the external world, even static, is not  
372 reducible to a vector with independent spatial and temporal coordinates (Pellionisz and Llinas,  
373 1982). It involves entangled flows of neural activity which are spatiotemporally distributed in  
374 the brain, from the retinal ganglion cells to the oculomotor neurons. Furthermore, the  
375 involvement of a measurable movement (like a saccade) is critical to guarantee that the  
376 associated neural activity corresponds to a real object and not to an illusion. The interceptive  
377 saccades have indeed been shown to be immune to strong visual illusions: they aim at the  
378 physical, not the perceived location of the target (Lisi and Cavanagh, 2015).

379         The neurophysiological characterization is also complicated by the changing location  
380 of the object. The time course of the motion can indeed look smooth and continuous, but such  
381 retrospective evaluation does not guarantee that the neural image follows a similar dynamics.  
382 In the case of tracking a moving target, we will see in the next section that the visuo-oculomotor  
383 brain does not spontaneously transform the smooth motion of a small object into a continuous  
384 smooth movement of the eyes. A complete dynamic morphing requires several days of training  
385 (Bourrelly et al., 2016a,b). Synchronizing the eye movements with the motion of a target is the  
386 outcome of a gradual process during which the brain activity assimilates the combination of  
387 visual and oculomotor signals. Contemporary habits of language and the Newtonian  
388 mechanics lead us to consider that an object is moving within a three- dimensional,  
389 homogeneous, isotropic and neutral “receptacle,” changing discretely its location in such a way  
390 that when it is here and now, it is not anymore where it was before, nor where it will soon be.  
391 In the brain activity corresponding to this object, there is no such thing as a “here and now”  
392 local point (Pellionisz and Llinas, 1982). The neural image obviously does not change as fast  
393 as its associated object in the external world; some residual activity persists “behind” (Goffart  
394 et al., 2016, 2017a; Keller et al., 1996). It also expands toward neighboring cells, driving the  
395 gaze toward locations in the external world where the target never went (Mitrani and Dimitrov,  
396 1978; Quinet and Goffart, 2015a). Given the complexity of the problem when one considers  
397 the neural image of a real object moving in front of our eyes, how do we address the problem

398 of a putative internal model without doing disappointing parodies of the neurophysiological  
399 processes and their oculomotor expression?

400 Because of all these complications, we will avoid considering the notion of internal  
401 model to understand how the brain endows the animals with the ability of maintaining the  
402 direction of their gaze toward the present location of the target, i.e., where it is here and now.  
403 Instead, our working hypothesis is that spatiotemporally accurate actions result from an  
404 adaptive process which consists in synchronizing the outcome of visuomotor flows of activity  
405 with the target dynamics.

## 406 **6. LEARNING TO TRACK THE CHANGING LOCATION OF A MOVING** 407 **TARGET**

408 A target moving in the visual field triggers an interceptive saccade that brings the target  
409 image onto the fovea. Then, this foveation is more or less efficiently maintained by two types  
410 of eye movements: low-velocity (pursuit) and high-velocity movements (catch-up saccades).  
411 Fleuriet and Goffart (2012) hypothesized that the oculomotor system is driven by an estimate  
412 of the expected current target location, and subsequent experiments indicated that this  
413 command is merging signals from the recent past with mnemonic signals carrying an  
414 expectancy built upon repeated experience.

415 It is indeed obvious that the ability to continuously encode, in spite of neural conduction  
416 and stabilization delays, the “here-and-now” location of a moving target, requires that its  
417 trajectory is already known. To further develop this hypothesis, we studied how inexperienced  
418 monkeys learned to track a target which always moved along the same path. During these  
419 experiments, each trial started with the foveation of a central target which, after a variable  
420 interval, stepped on the vertical meridian (upward or downward) and immediately after, moved  
421 horizontally with a constant, increasing or decreasing velocity (Bourrelly et al., 2016a,b). The  
422 target moved toward the left or the right for 800 ms before disappearing. The monkey was  
423 rewarded if, after making a saccade in the appropriate visual quadrant, it tracked the target  
424 until its ultimate location. During the first days, the tracking was composed of saccades  
425 separated by periods during which gaze moved slowly in the same direction as the target.  
426 Except for the brief periods during which fairly accurate saccades occurred, the gaze lagged  
427 behind the target most of the time.

428 < **Figure 5 approximately here** >

429 Fig. 5 illustrates qualitatively with example trials recorded in five monkeys, how the  
430 oculomotor tracking changed between the first and last sessions of the training procedure,  
431 when the target moved with a constant speed. During the first session (left column), after the  
432 interceptive saccade, the eye followed the target with a velocity which was not sufficient to  
433 maintain its image on the fovea. The velocity of the postsaccadic movement did not match the  
434 target speed. Contrary to the experiments where the target moved with an increasing speed  
435 (Fig. 6), the eye did not increase the velocity of its movement before the subsequent catch-up  
436 saccade. After several daily training sessions (right column), the monkey was able to smoothly  
437 track the target immediately after the interceptive saccade; the catch-up saccades either  
438 disappeared or their amplitude was largely diminished. This saccade-free tracking was rarely,  
439 if ever, observed during the first session. The reader will find in Bourrelly et al. (2016a) a  
440 detailed description of how tracking eye movements changed during the course of one trial,  
441 during the course of the first and last sessions and across the successive sessions. Fig. 6  
442 illustrates the same evolution when an accelerating target is tracked (Bourrelly et al., 2016b).  
443 After the interceptive saccade, contrary to the experiments which tested the tracking eye  
444 movements made in response to a target moving with a constant speed, the postsaccadic eye  
445 movements could accelerate when the target accelerated, but not sufficiently to match the  
446 increasing target speed. With repeated practice, i.e., with a larger number of trials and over  
447 the consecutive days, the pursuit gain (ratio of eye to target velocities during the intersaccadic  
448 intervals) enhanced while the number and amplitude of catch-up saccades diminished  
449 (Bourrelly et al., 2016a). The evolution of tracking from an intermittent to a continuous “here-  
450 and-now” mode is actually inevitable because the saccades exert a facilitative influence on  
451 pursuit eye movements. This facilitation is twofold, visual and motor. On the one hand,  
452 saccades bring the target image within the central visual field, where the visual motion signals  
453 are more effective for driving slow eye movements compared with signals from the peripheral  
454 field (Lisberger and Westbrook, 1985). On the other hand, the slow eye movements match the  
455 target speed better when they are preceded by a saccade (Lisberger, 1998). We will see in the  
456 next section the neurophysiological process which possibly underlies this facilitation.

457 < **Figure 6 approximately here** >

## 458 **7. NEURAL BASIS OF SACCADIC AND PURSUIT EYE MOVEMENTS**

459 The gaze direction is determined by the cooperative action of six muscles attached to  
460 each eyeball. These muscles are called extraocular muscles, to distinguish them from those  
461 located inside the eye and which influence the diameter of the pupil or the lens curvature. Two  
462 pairs of muscles are involved during horizontal rotations of the eyes (i.e., rotations about a

463 vertical axis through the eyeball): the lateral rectus (LR) and medial rectus (MR) muscles.  
464 Orienting gaze toward the right is accomplished in the right eye by the contraction of the LR  
465 and the relaxation of the MR, while in the left eye the MR is contracted and the LR relaxed.  
466 Conversely, orienting gaze toward the left is produced in the left eye by the LR contraction and  
467 MR relaxation concomitantly with the MR contraction and the LR relaxation in the right eye.  
468 Vertical movements of the eyes (rotations about a horizontal axis) are slightly more complex  
469 than the horizontal ones because they are the result of a synergy between the other four  
470 extraocular muscles. The joint contraction of the superior rectus (SR) and inferior oblique (IO)  
471 muscles of both eyes, combined with the relaxation of the inferior rectus (IR) and superior  
472 oblique (SO) muscles, rotate the eyes upward. The bilateral contraction of the IR and SO,  
473 combined with the relaxation of SR and IO, rotates them downward. Oblique saccades are  
474 generated by tensions simultaneously developed by the muscles rotating the eye horizontally  
475 and those rotating the eye vertically. The contraction of extraocular muscles is driven by the  
476 activity of MNs located in cranial nerve nuclei III (oculomotor), IV (trochlear), and VI  
477 (abducens).

478 Different populations of premotor neurons in the reticular formation are involved in the  
479 generation of saccadic and slow eye movements. The generation of saccadic eye movements  
480 involves burst discharges by neurons located in the pontomedullary and mesencephalic  
481 reticular formation, respectively, for the horizontal and vertical movements (see Goffart, 2017  
482 for a brief review and references). Because they emit a burst of action potentials within a very  
483 short-time interval, these neurons have been called saccade-related burst neurons. Although  
484 it can be preceded by a prelude firing, it is the burst which drives the MNs and yields the rapid  
485 rise in the tension of extraocular muscles during saccades. Lesions of the regions where the  
486 burst neurons are located abolish the ability to generate saccades without compromising the  
487 ability to make slow eye movements toward a moving target (Henn et al., 1984). The  
488 generation of pursuit eye movements involves other groups of neurons called tonic neurons  
489 which are located in distinct nuclei of the reticular formation (Sharpe, 2008).

490 A dissociation of the neural processes leading to the generation of saccadic and pursuit  
491 eye movements is supported by multiple clinical and experimental lesion studies (e.g., Büttner  
492 et al., 1994; Henn et al., 1984; Morrow and Sharpe, 1995; Ohtsuka et al., 1992; Pierrot-  
493 Deseilligny et al., 1990; Sharpe, 2008). However, this segregation is not absolute. A coupling  
494 between the processes controlling each type of movement is indeed suggested by the  
495 existence of neurons which modulate their sustained firing rate during both types of movement.  
496 Most of these neurons have been found in the cerebellum, and more specifically in its floccular  
497 and parafloccular regions (Noda and Mikami, 1986; Noda and Suzuki, 1979) and in its



498 medioposterior territories, i.e., the caudal fastigial nuclei (Fuchs et al., 1994) and the posterior  
499 vermis (Suzuki and Keller, 1988). Sato and Noda (1992) also found Purkinje cells in the  
500 posterior vermis which fire during saccades and pursuit eye movements, but these cells seem  
501 to be rare since the activity of only 6.6% (44 units out of 663) was modulated during pursuit  
502 eye movements. Of these units, less than 4% were located in the oculomotor vermis (OMV).  
503 This last point is important because it led the authors to conclude that the OMV did “not qualify  
504 as a structure responsible for the control of smooth pursuit,” a conclusion which contrasts with  
505 the recent claim that “the overwhelming majority [81%] of Purkinje cells in the OMV are ‘dual’  
506 PCs responding to both types of goal-directed eye movements” (Sun et al., 2017). Suzuki and  
507 colleagues were the first to report neurons which, in the posterior vermis, modulate their firing  
508 rate during both saccadic and slow eye movements (Suzuki et al., 1981). They report that “the  
509 nature and number [of saccade-related activities in the posterior vermis] indicate that there is  
510 only a slight overlap between the population of smooth-pursuit and vision-related units and the  
511 population of saccade-related cells previously observed in lobules VI and VII.” The observation  
512 that large lesion in the posterior vermis impairs saccadic and pursuit eye movements (Takagi  
513 et al., 1998, 2000) does not exclude the possibility of two or three segregated groups of  
514 neurons. Therefore, the role of the medioposterior cerebellum in the control of saccadic and  
515 slow eye movements requires further investigation. In the brainstem, cells modulating their  
516 firing rate during saccades and pursuit have also been reported, notably in the rostral part of  
517 the SC (Krauzlis, 2001) toward which the nucleus prepositus hypoglossi and the caudal  
518 fastigial nuclei project (Hartwich-Young et al., 1990; May et al., 1990), as well as in the nucleus  
519 raphe interpositus (RIP) where pause neurons are located. The two rostral SC participate in  
520 adjusting oculomotor commands for maintaining a target within the central visual field (Goffart  
521 et al., 2012; Hafed and Krauzlis, 2008; Hafed et al., 2008), a situation which is often  
522 encountered when a moving target is being pursued. Concerning the pause neurons, it is  
523 commonly believed that they maintain gaze steady during fixation by gating the burst of  
524 saccade-related premotor neurons. Unfortunately, this idea will remain speculative as long as  
525 the study of its inactivation has not demonstrated the presence of irrepressible saccades during  
526 delayed saccade tasks, i.e., when a peripheral target competes with a central one. The  
527 observation that pause neurons reduce their firing during the initiation of pursuit eye  
528 movements led Missal and Keller (2002) to imagine an inhibitory role of these neurons in  
529 pursuit. Unfortunately here again, this option requires further investigation because the  
530 reduced firing could merely signal the preparation of a catch-up saccade toward the moving  
531 target. Additional studies of the effects of RIP inactivation are also required to identify and  
532 characterize the neural networks to which they belong.

## 533 **8. ENLARGEMENT OF THE NEURONAL MASS FOR ACCURATE TRACKING**

534 Beyond these unknowns, a general neurophysiological process can explain the  
535 changes of the saccadic and slow components of oculomotor tracking during repeated  
536 exercise. We propose that they result from an expansion of the “mass” of neural activity linked  
537 to the visuomotor representation of the central field. Several empirical facts converge to  
538 support this hypothesis. First, it is a basic fact that the saccades orient the fovea toward  
539 locations situated next to the moving target (e.g., Fig. 3). As a result, neurons which are  
540 sensitive to the visual stimulation of the fovea become excited by the presence of the target.  
541 When the target is moving, the repeated excitation of these neurons by the catch-up saccades  
542 likely contributed to delay the adaptation of their response, and the variability of saccade  
543 endpoints enlarged the population of active neurons. Such an enlargement would functionally  
544 correspond to an expansion of the central visual field, which is also known to involve neurons  
545 in the rostral SC. Thus, the increase in pursuit velocity and the reduction of saccade size  
546 observed across the learning sessions (Bourrelly et al., 2016a) could result from an  
547 enlargement of the population of active neurons in the rostral portion of the SC. The  
548 rostral SC has indeed been shown to be involved in both foveal and parafoveal pursuit (Basso  
549 et al., 2000; Hafed et al., 2008; Krauzlis et al., 2000) as well as during fixational saccades  
550 (Hafed and Krauzlis, 2012). The parallel facts that a larger target size not only improves the  
551 accuracy of pursuit eye movements (Helmick, 1951; Pola and Wyatt, 1985) but also increases  
552 the spatial spread of activity in the rostral SC (Goffart et al., 2012; Hafed et al., 2008) are  
553 consistent with this hypothesis of enlarged population.

554 This enlargement could be under the influence of the caudal fastigial nuclei, though  
555 nothing excludes the participation of other deep cerebellar nuclei. Concerning the cFN, their  
556 pharmacological inactivation is known to impair the generation of pursuit and saccadic eye  
557 movements (Bourrelly et al., 2017; Buzunov et al., 2013; Goffart et al., 2004; Robinson et al.,  
558 1997), as well as the direction of gaze relative to a fixated target, whether the latter is static  
559 (Guerrasio et al., 2010) or moving (Bourrelly et al., 2017; Robinson et al., 1997). This gaze  
560 “positional” disorder (called fixation offset) has been proposed to involve the fastigio-collicular  
561 connection (Guerrasio et al., 2010; May et al., 1990) because similar fixation offsets are  
562 observed in the head-restrained monkey during unilateral inactivation of rostral SC (Goffart et  
563 al., 2012; Hafed et al., 2008). But the fastigial involvement in generating accurate pursuit eye  
564 movements is not restricted to the fastigio-collicular channel; projections to precerebellar nuclei  
565 such as the nucleus reticularis tegmenti pontis or dorsal pontine nuclei have been documented  
566 (Batton et al., 1977; Noda et al., 1990; Sugita and Noda, 1991). These nuclei constitute major  
567 relays by which motion signals reach the flocculus and paraflocculus for the generation of

568 pursuit eye movements (Belton and McCrea, 1999; Langer et al., 1985; Mustari et al., 2009;  
569 Zee et al., 1981). There again, an enlargement of the population of active neurons could also  
570 happen under the control of caudal fastigial nuclei (as recently demonstrated for the control of  
571 the saccade size; Goffart et al., 2017b; see also Quinet and Goffart, 2015b) and possibly also  
572 of other deep cerebellar nuclei (Robinson and Fuchs, 2001). More fundamentally, regardless  
573 of the targeted neural region, an increase of the number of active neurons enhances the  
574 probability of synchronous action potentials which, as explained in the second section of this  
575 chapter, reduces the transmission delays. Thus, motion signals reach faster the tonic neurons  
576 for driving the slow movements of the eyes when a moving target is being tracked.

## 577 **9. CONCLUSION**

578 In this chapter, a new line of investigation has been proposed to identify the  
579 neurophysiological processes which underlie the ability to produce goal-directed eye  
580 movements which are accurate, spatially and temporally. We have shown that tracking the  
581 location (here and now) of a moving target is not necessarily and spontaneously smooth, that  
582 such smoothness is in fact the consequence of training. Because of the visuomotor delays, the  
583 ability to generate eye movements which are locked onto the current target location is  
584 obviously not possible unless the upcoming trajectory is already known. Several studies have  
585 shown that the velocity of slow eye movements hardly match the target speed when an  
586 unpredictable target is being tracked (e.g., Bahill et al., 1980; Barnes and Collins, 2015; Deno  
587 et al., 1995; Miall et al., 1986; Michael and Melvill-Jones, 1966; Mrotek et al., 2006; St-Cyr and  
588 Fender, 1969). The presence of numerous catch-up saccades makes the tracking look  
589 intermittent. Moreover, the absence of lead between the gaze and target directions and the  
590 frequent lags tell us that the visuomotor brain works with a very short horizon of predictability  
591 during visual tracking. Without assuming the existence of “predictive” signals or any encoding  
592 of future target locations in the brain activity, we have explained how the spatial  
593 synchronization is largely facilitated by the generation of saccades. Finally, considering that  
594 “low gain smooth pursuit is a sensitive sign of chronic diffuse brain disease that can be useful  
595 in the detection of neurological disorders” (Sharpe, 2008), for those disorders where the action  
596 is spatiotemporally detached from the current context, the rehabilitation of neurological patients  
597 may be facilitated by the development of strategies to enhance their visuomotor presence.

598

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612

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## 859 **FURTHER READING**

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866 **FIGURE LEGENDS**

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868

869 Figure 1: Effect of target contrast on the latency of saccades. Time course of the horizontal  
870 component of saccades aimed at a static target LED located 8 to the right in a dimly illuminated  
871 room. A gap interval separated the fixation of a central target from the onset of the brief  
872 peripheral target (50 ms duration). Additional methodological information can be found in  
873 Goffart et al., (2006).

874

875 Figure 2: Saccades evoked by electrical microstimulation of one site in the deep superior  
876 colliculus. A: Reducing the rate of stimulation pulses (from 500 to 300 pulses per second)  
877 increases the latency of evoked saccades. B: The saccades (same as in A) have identical  
878 velocity profiles. C: Reducing further the rate of stimulation pulses (from 300 to 200 pulses per  
879 second) made the latencies of evoked saccades even longer and more variable. D: Increasing  
880 the current of stimulation reduces the latency of saccades.

881

882 Figure 3: Comparison of targeting errors between saccades toward a static versus a moving  
883 target. During the static target trials, the target appeared at locations comparable to those  
884 travelled by the moving target. The error is plotted as a function of the duration of the largest  
885 saccade component, i.e., the component orthogonal to the motion (when the target is moving).  
886 In A, the target moved horizontally toward the left or the right, from locations situated 8 or 16  
887 degrees along the vertical meridian. In B, the target moved vertically upward or downward,  
888 from locations situated 8 or 16 degrees along the horizontal meridian. Negative values  
889 correspond to a hypometria (undershoot). Data collected in one monkey (N). Additional  
890 information can be found in Fleuriet et al., (2011).

891

892 Figure 4: Time course of horizontal and vertical eye movements toward a temporarily invisible  
893 target. The target moved horizontally away from the vertical meridian, to the left (A,C) or to the  
894 right (B,D), in the upper (top) or lower (bottom) visual field, and with a constant speed (20°/s).  
895 After a short interval of 150 ms, the target disappeared during 300 ms (grey zone) and  
896 reappeared at the location where it would have been if always visible, moving at the same  
897 speed. The black traces correspond to control trials, the blue traces to perturbed trials. During  
898 these trials, the trajectory of interceptive saccades was perturbed by a brief electrical  
899 microstimulation (30 ms, 12μA, 400 pps) in the deep superior colliculus. The black arrows  
900 indicate the electrically-evoked saccade (perturbation). The green and blue arrows indicate  
901 catch-up saccades made while the target is invisible during control and perturbed trials,  
902 respectively. Additional methodological information can be found in Fleuriet and Goffart (2012).

903

904 Figure 5: Typical oculomotor behaviour of a monkey tracking a visual target moving horizontally  
905 with a constant speed. The horizontal eye position is plotted as a function of time after the  
906 target motion onset for three trials recorded during the first (left column: Beginning) and last  
907 training sessions (right column: End). The time course of horizontal target position is illustrated  
908 by the red line. The selected trials were recorded in 5 monkeys (A, B, C, M and G: from top to  
909 bottom, respectively) when the target moved in the upper right quadrant with a constant speed  
910 ( $20^\circ/\text{s}$ ). During the other randomly interleaved trials, the target moved similarly, horizontally  
911 and away from the vertical meridian, but in the lower right, the lower left or the upper left  
912 quadrant. Additional methodological information can be found in Burrelly et al. (2016a).

913

914 Figure 6: Typical oculomotor behaviour of a monkey tracking an accelerating visual target. The  
915 horizontal eye position is plotted as a function of time after the target motion onset for three  
916 trials recorded during the first (left column: Beginning) and last training sessions (right column:  
917 End). The time course of horizontal target position is illustrated by the red line. The selected  
918 trials were recorded in 5 monkeys (A, B, C, M and Bi: from top to bottom, respectively) when  
919 the target moved horizontally in the upper right quadrant with a speed which increased from 0  
920 to  $40^\circ/\text{s}$  during 800ms. During the other randomly interleaved trials, the target moved similarly,  
921 horizontally and away from the vertical meridian, but in the lower right, the lower left or the  
922 upper left quadrant. The experimental protocol is identical to that described in Burrelly et al.  
923 (2016a).

Figure 1  
Goffart, Bourelly and Quinet *Prog Brain Res* 2017

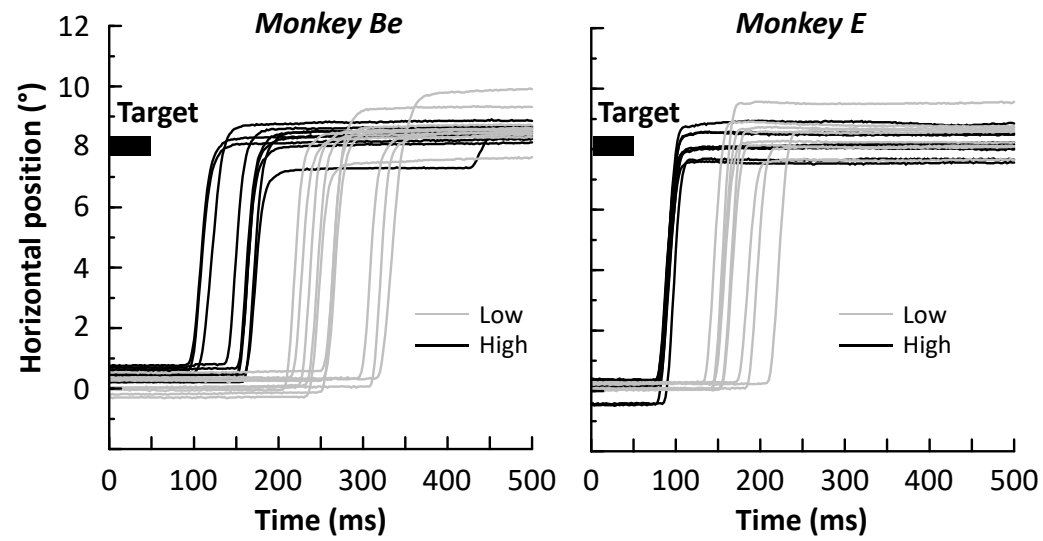


Figure 2  
Goffart, Bourelly and Quinet *Prog Brain Res* 2017

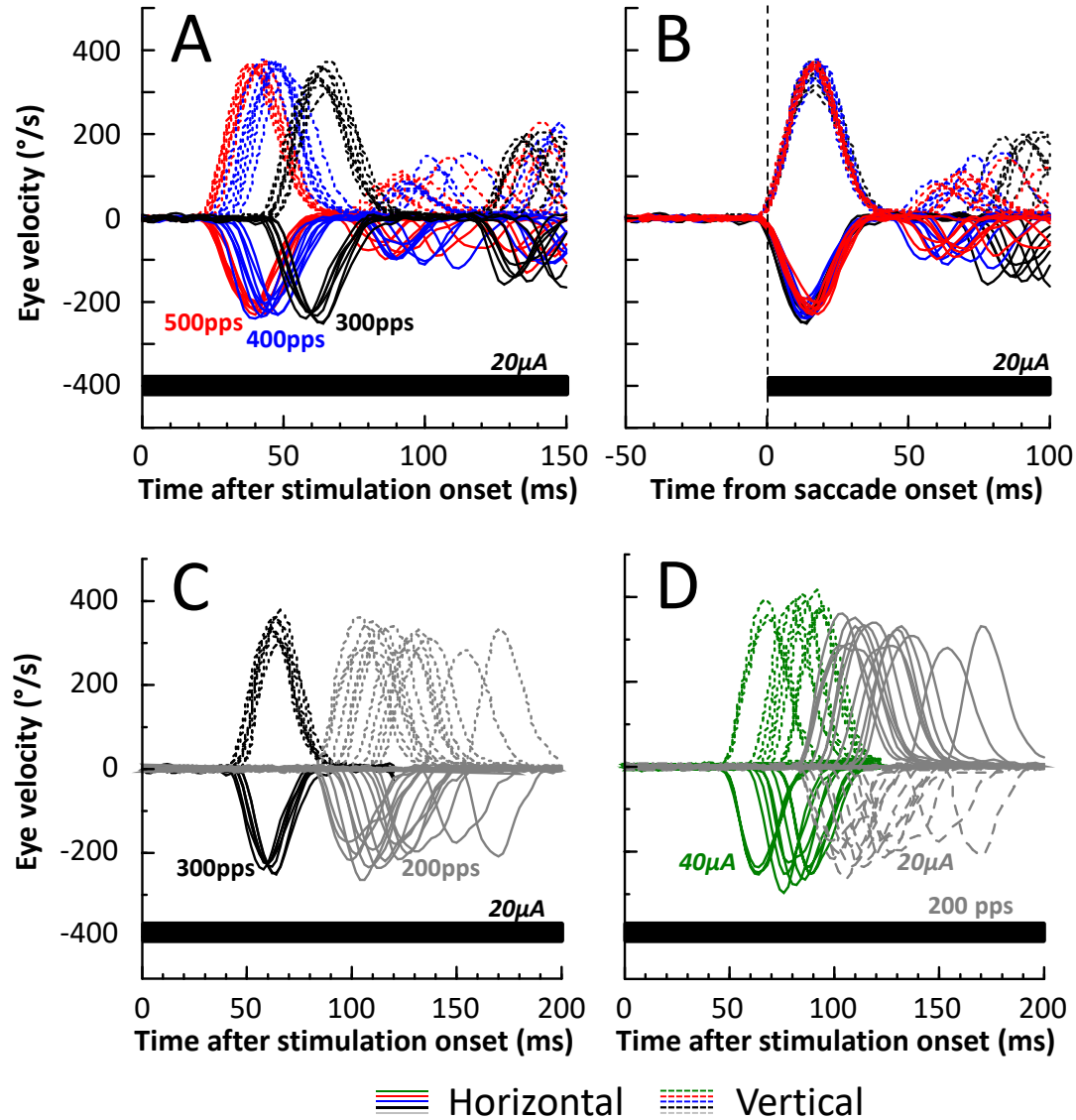


Figure 3  
Goffart, Bourelly and Quinet *Prog Brain Res* 2017

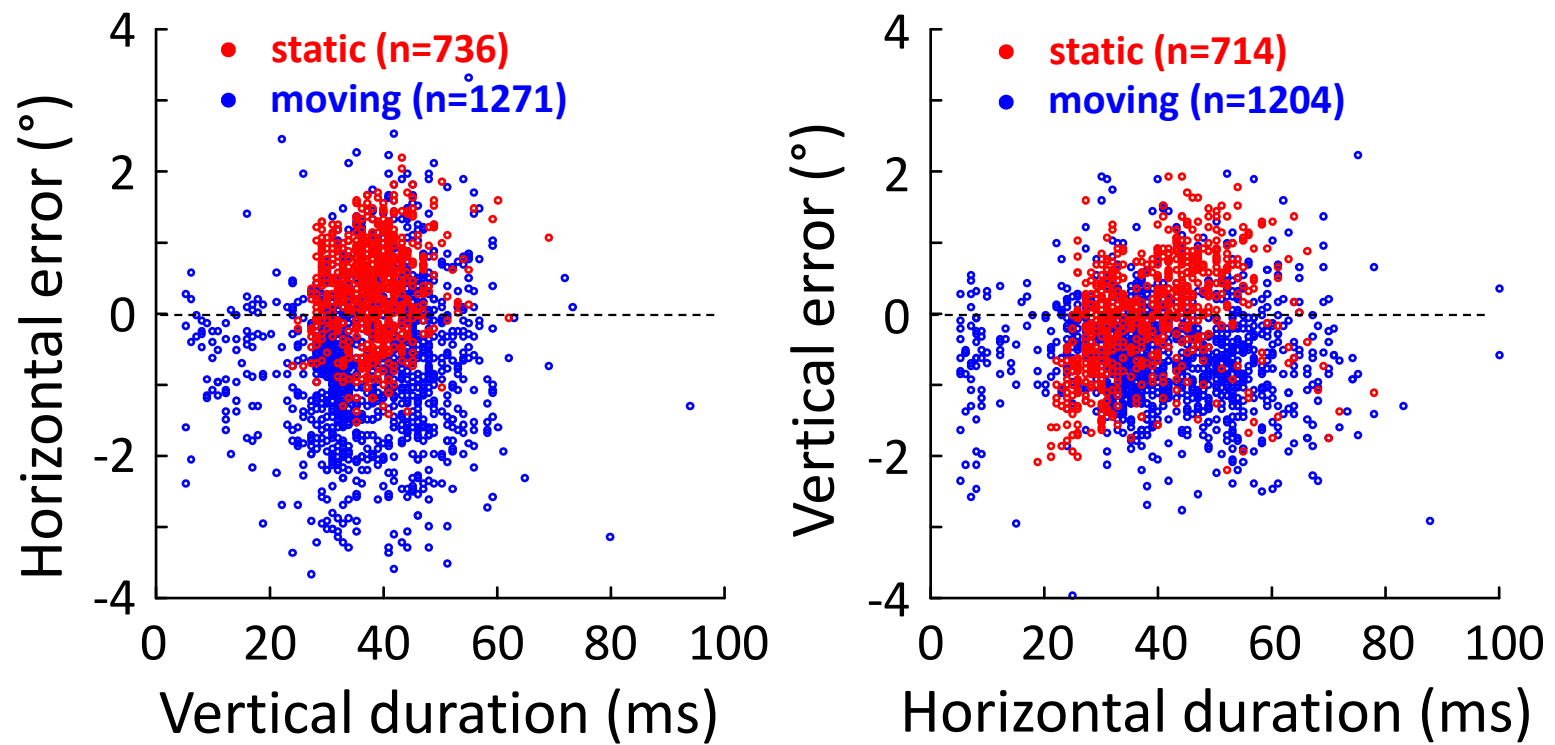
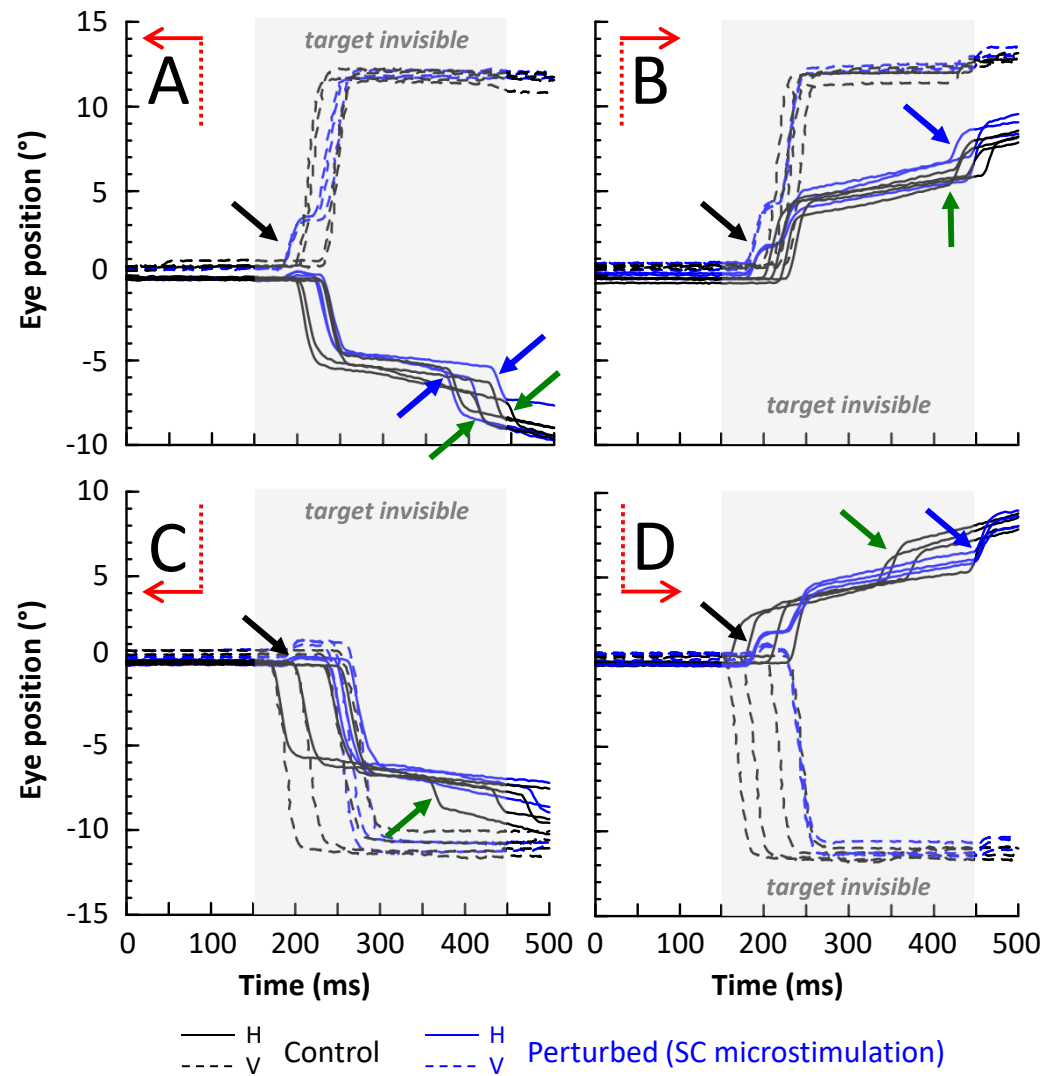




Figure 4  
Goffart, Bourelly and Quinet *Prog Brain Res* 2017



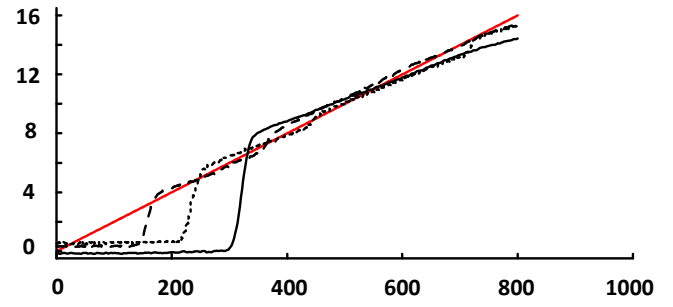
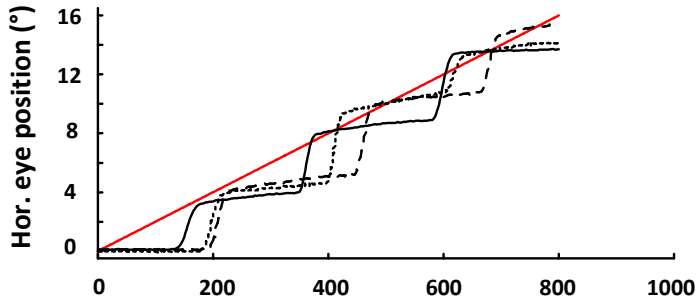
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Figure 5

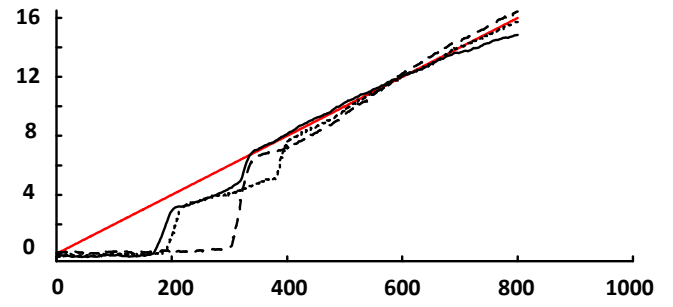
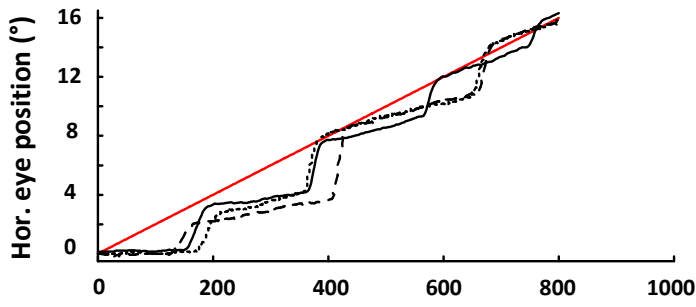
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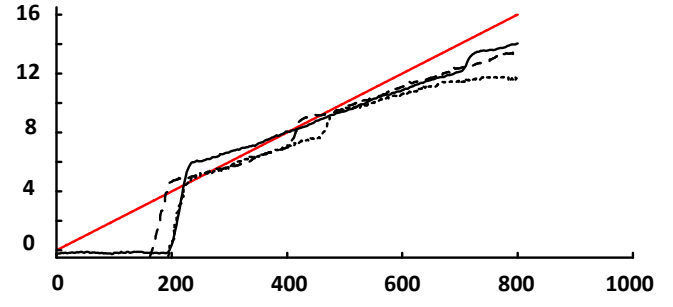
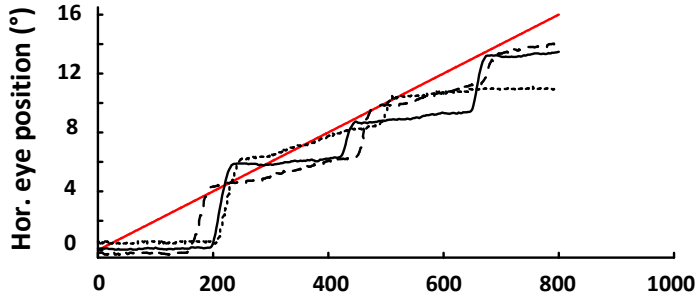
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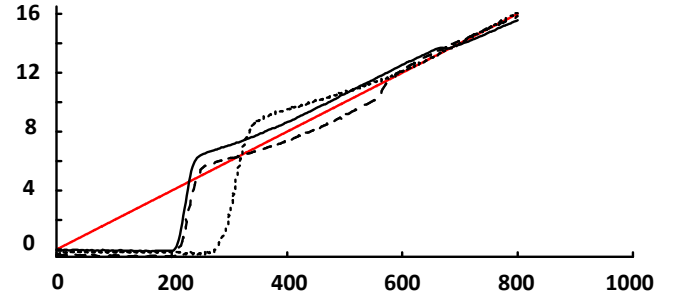
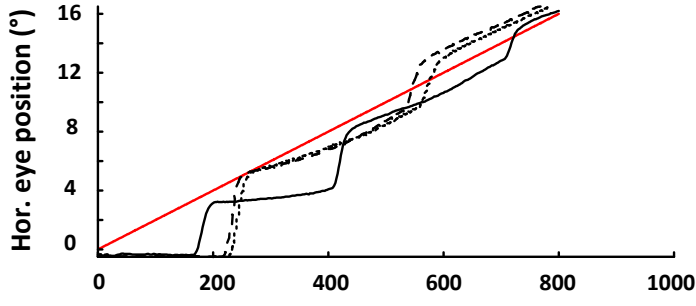
*Monkey B*



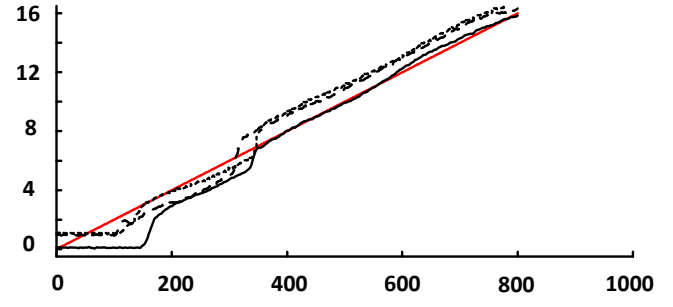
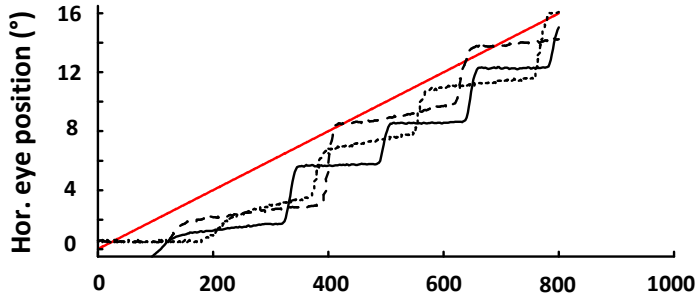
*Monkey C*



*Monkey M*



*Monkey G*



Time (ms)

Time (ms)

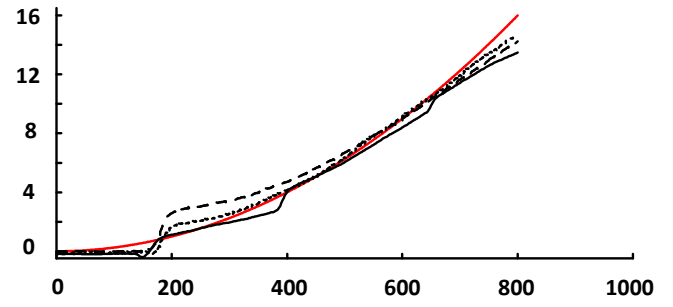
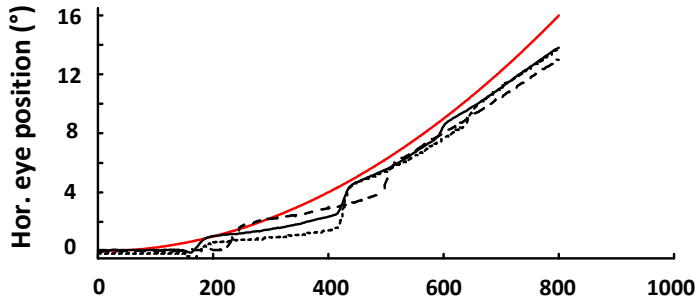
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Figure 6

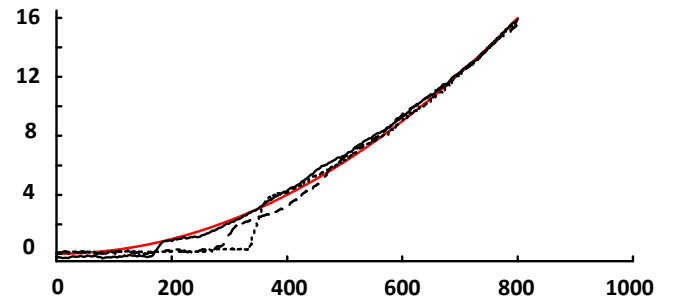
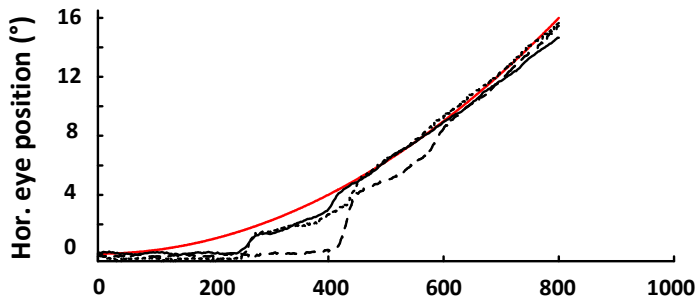
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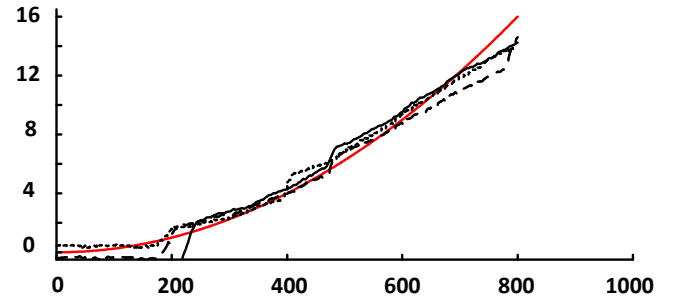
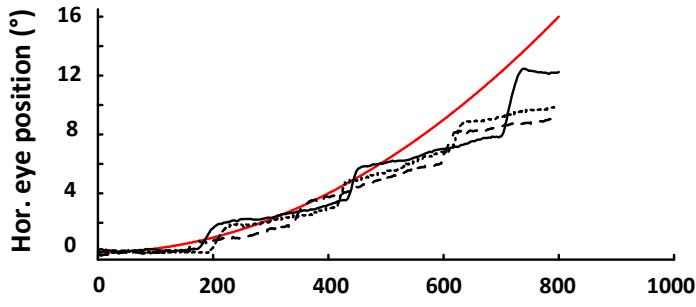
*Monkey A*



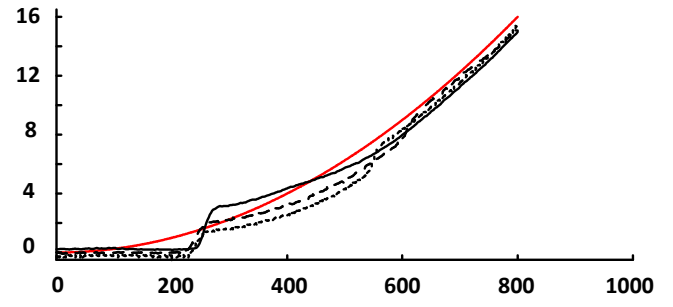
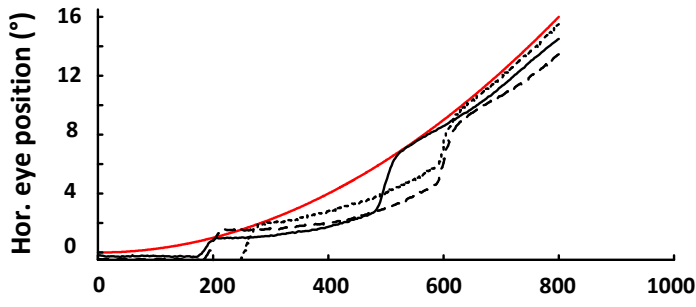
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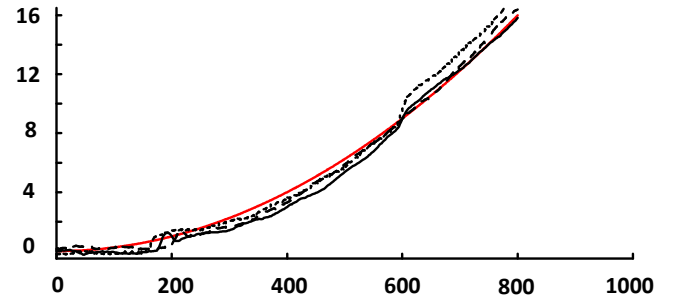
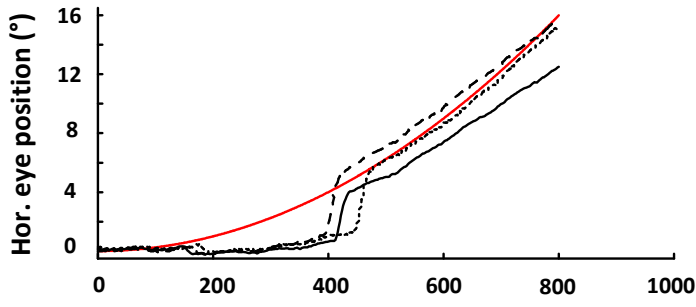
*Monkey C*



*Monkey M*



*Monkey Bi*



Time (ms)

Time (ms)