



**HAL**  
open science

# Kinematics and the neurophysiological study of visually-guided eye movements

Laurent Goffart

► **To cite this version:**

Laurent Goffart. Kinematics and the neurophysiological study of visually-guided eye movements. Mathematical modelling in motor neuroscience: State of the art and translation to the clinic, 2019, 10.1016/bs.pbr.2019.03.027 . hal-02109608

**HAL Id: hal-02109608**

**<https://hal.science/hal-02109608>**

Submitted on 24 Jun 2020

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

1 To be published in: *Progress in Brain Research* volume 248, chapter 68

2

3 Kinematics and the neurophysiological study of visually-  
4 guided eye movements

5

6 Laurent Goffart<sup>1,2</sup>

7

8 Number of words:

9

abstract: 133

10

text + references: 4063

11

legend: 111

12

13 1. Aix Marseille Univ, CNRS, INT, Inst Neurosci Timone, Marseille, France

14 2. Aix Marseille Univ, CNRS, CGGG, Centre Gilles Gaston Granger, Aix-en-Provence, France

15

16 Address for correspondence: laurent.goffart@univ-amu.fr

17

18 **ABSTRACT** (134 words):

19 Notions of kinematics have been used to “decode” the firing rate of neurons and to explain  
20 the neurophysiology underlying the generation of visually-guided eye movements. The  
21 appropriateness of their fitting to events occurring within a medium (the brain) radically  
22 different from the physical world was not questioned. Instead of embedding the eye  
23 kinematics in the firing rate of central neurons, we propose that the saccadic and pursuit eye  
24 movements in fact reflect the dynamics of transitions of brain activity, from unbalanced  
25 states to equilibrium (symmetry) between directional tendencies carried by the recruited  
26 visuomotor channels, with distinct transitions characterizing each movement category.  
27 While the eyeballs conform to the physical laws of motion, the neural processes leading to  
28 their movements follow principles dictated by the intrinsic properties of the brain network  
29 and of its diverse neurons.

30

31 **KEYWORDS:**

32 Tracking, saccade, pursuit, foveation, symmetry, equilibrium, neurophysiology, kinematics

33

34 **ACKNOWLEDGMENTS**

35 The author thanks Professors Gabriella Crocco and Igor Ly for epistemological advices.

36

## 37 **1. GAZE, TARGET AND MEASUREMENTS.**

38           During the characterization of the neuronal processes which underlie the ability to  
39 visually capture a moving object, the neurophysiological studies have been led to embed  
40 within the inner functioning of the brain, notions which belong to the vocabulary of  
41 kinematics. When they rotate in the orbit, the eyeballs do not change their shape, and this  
42 rigidity allows specifying their orientation with a few numbers (coordinates) whose values  
43 depend upon the physical reference frame that has been chosen. Thus, gaze became  
44 assimilated to a line (the line of sight) or to an axis (visual axis), shifting from one point in the  
45 visual field to another, even though gaze actually refers to an extended field of binocular  
46 visuomotor interactions (Hafed et al., 2015; Krauzlis et al., 2017; Otero-Millan et al., 2014).

47           Attributing point-like values to gaze and targets inevitably leads to numerical  
48 differences between them, especially when their measurement is made with higher  
49 resolution. However such differences between measured values should not lead us to think  
50 that corresponding differences necessarily exist within the brain functioning. Objects in the  
51 physical world are obviously not points and during visual fixation, all light beams do not  
52 converge onto one single photoreceptor. The numerical values attributed to gaze and target  
53 directions belong to a “medium” which is not the brain but the set of behavioral  
54 measurements. Within the medium of brain functioning, a target, whether it is located in the  
55 central or peripheral visual field, is not point-like. From the retinal ganglion cells to their  
56 post-synaptic targets, and from the latter to their targets in the cerebral cortex or the  
57 brainstem, the divergence of anatomical projections implies that the neuronal image of any  
58 physical object, even tiny, recruits an increasing number of neurons (e.g., Nowak and Bullier,  
59 1997).

60           When we record the activity of neurons in cortical visual areas, we find that they emit  
61 action potentials whenever a stimulus appears within a more or less extended region of the  
62 visual field. Likewise, when we record neurons in saccade-related regions (e.g., deep  
63 superior colliculus), we discover bursts of action potentials whenever a saccade is made  
64 toward locations situated within a more or less bounded region of the visual field. The  
65 extent of response fields indicates that any visual object, or any saccade toward its location,  
66 involves a large set of neurons (McIlwain, 1976; Sparks et al., 1976). Moreover, in most  
67 visual and saccade-related regions, the neurons are laid out in such a way that neighboring  
68 neurons respond to neighboring stimuli in the visual field, or burst during saccades toward  
69 neighboring locations. Despite the divergence of anatomical projections and the lateral  
70 extent of post-synaptic contacts, the retinal topology is preserved across the layer of  
71 neurons.

72           The consequence of these two basic observations is that neighboring objects in the  
73 visual field, or saccades toward their respective location, mobilize neuronal assemblies  
74 which involve common cells. This overlap is not taken into account when the focus is made  
75 upon the numerical difference between gaze and target directions, the value of which is  
76 called *position error* or *motor error*. Such an error signal is indeed considered as the critical  
77 factor triggering a saccade and specifying its metrics (amplitude and direction).

## 78 **2. GAZE DIRECTION AS EQUILIBRIUM.**

79           The simplest solution that was proposed to model the execution of saccades is a  
80 negative feedback loop reducing this difference, i.e., between a desired direction of gaze and  
81 an estimate of its current direction. Desired gaze direction corresponds to the selected  
82 target location. The *motor error* resulting from the comparison between these two estimates

83 would feed the premotor neurons which themselves would emit action potentials at a rate  
84 proportional to the size of the error (van Gisbergen et al., 1981). Thus, while the gaze  
85 direction moves toward the target, the motor error diminishes and the firing of premotor  
86 neurons declines and cease exciting the motor neurons when the error has been zeroed  
87 (Robinson, 1975).

88 In this theoretical framework, a non-zero motor error leads to a saccade. However,  
89 recent studies in the monkey reported pathological cases where stable fixation is engaged  
90 although gaze is not directed toward the location which was previously fixated during non-  
91 pathological (control) conditions. Gaze is directed toward a location which is offset with  
92 respect to the target; no saccade is launched in spite of non-zero motor error. For example,  
93 when the activity of caudal fastigial nuclei (cFN) is altered by a local and unilateral injection  
94 of a pharmacologically-inhibiting agent (muscimol), the monkey does not direct its gaze  
95 toward the target (located straight ahead) but toward a location slightly shifted toward the  
96 injected side (Ohtsuka et al., 1994; Goffart et al., 2004; Guerrasio et al., 2010; Robinson et al.  
97 1993). By contrast, when the injected drug is a disinhibiting agent (bicuculline), the gaze is  
98 deviated toward the opposite side (Sato and Noda, 1992). The ipsilesional fixation offset  
99 observed after muscimol injection is not an oculomotor disorder because when the head is  
100 free to move, the monkey exhibits an ipsilesional deviation of the head (cervical dystonia)  
101 and the eyes in the orbit are deviated toward the contralesional side (Quinet and Goffart,  
102 2005). The relatively similar sizes of fixation offset between the head-restrained and –  
103 unrestrained testing conditions suggest an alteration of processes underlying gaze  
104 orientation. This deduction is consistent with anatomical studies which report cFN  
105 projections to the rostral parts of both SC (May et al., 1990) and not to the nuclei prepositus  
106 hypoglossi (NPH) or medial vestibular nuclei (MVN) where tonic neurons projecting to

107 abducens motor neurons are found (Noda et al., 1990). In the rostral SC, the neurons fire in  
108 a sustained manner when the same gaze direction is maintained (Dorris and Munoz, 1995),  
109 but they also emit bursts of action potentials during fixational saccades (Hafed and Krauzlis,  
110 2012). When muscimol is injected in this part of the SC, the monkey exhibits a fixation offset  
111 when it directs its gaze toward a straight ahead target. By contrast, saccades to peripheral  
112 targets are accurate (Goffart et al., 2012). A fixation offset has also been reported after  
113 muscimol injection in the frontal eye field (Dias and Segraves, 1999), a region which is  
114 reciprocally connected with the deep superior colliculus (Sommer and Wurtz, 1998).

115         These observations stress not only the limitations brought by reducing gaze and  
116 target to their measured values, but also the problem brought by assimilating their  
117 difference as a command leading to a saccade. When we consider gaze direction as the  
118 outcome of flow of signals propagating from the optic to the motor nerves, along multiple  
119 parallel channels involving neuronal assemblies distributed bilaterally in the brain, we are  
120 led to a viewpoint where a change in gaze orientation is not initiated when the flows involve  
121 omnidirectional commands that counter-balance each other (figure 1; Goffart et al., 2018).

### 122 **3. TRANSFORMING THE LOCATION OF A PERIPHERAL TARGET INTO** 123 **SACCADE DURATION.**

124         The concept of negative feedback loop was a simple solution to the fundamental  
125 problem of understanding how the *locus* of activity elicited by a visual target, in the retina or  
126 in the superior colliculus, is transformed into *duration* of motoneurons' bursting activity  
127 (Moschovakis et al., 1996; Scudder et al., 2002; Sparks, 2002). The solution was simple  
128 because it removed the need to search, within the brain activity, a process encoding saccade

129 duration, as Hans Kornhüber (1971) initially proposed, but later revisited (Jürgens et al.,  
130 1981). With the negative feedback control, a cerebral “chronometer” is not needed; the  
131 movement duration is a secondary by-product of a process reducing a difference between  
132 spatial magnitudes putatively encoded in the brain activity. This conceptual framework was  
133 fertile because it stimulated the making of multiple experiments which brought several new  
134 observations compatible with a negative feedback control. However, if the proposed signals  
135 (encoding of eye and target directions or displacements) and processes (comparator or  
136 resettable integrator) have no neurophysiological substrate, then the theory becomes  
137 irrefutable because it is impossible to demonstrate that something does not exist.

138         Later, on the basis of neuromimetic modeling, it was suggested that the signals used  
139 by models would not be explicitly conveyed by separate groups of neurons, but would  
140 correspond to activities involving assemblies of interconnected neurons distributed over  
141 several territories (Robinson, 1992; Optican and Quaia, 2002). Consequently, the signals  
142 imagined by the models are not tractable anymore with classical unit recording techniques.  
143 While the feedback control hypothesis encountered these complications, the hypothesis of  
144 Kornhüber (1971) was further developed by the group of Peter Thier (2011). Unfortunately,  
145 evidence for chronometric control was weak (see Goffart et al., 2017a; 2018 for  
146 explanations).

#### 147 **4. TRACKING A MOVING TARGET**

148         The notion of negative feedback control has also been used to explain the guidance  
149 of eye movements made when a subject tracked a moving visual target. Two processes  
150 would operate in parallel before the target foveation: one process would reduce the  
151 difference between gaze and target directions (same as discussed above) while the other

152 would reduce the difference between the eye and target velocities. Recording techniques  
153 indeed allow measuring eye movements with such high resolution that instantaneous  
154 velocity and acceleration can be calculated. When the performance of a subject tracking a  
155 moving target is measured, numerical differences between kinematic parameters of the eye  
156 and target can be calculated. Thus, notions such as “velocity error” and “acceleration error”  
157 were added to the list of stimuli driving the generation and execution of pursuit eye  
158 movements (Lisberger et al., 1987).

159           However, proposing that the instantaneous velocity of the eyeball (or of any physical  
160 object) is a parameter embedded within the inner functioning of the brain is a hypothesis  
161 which is difficult to take root in the neurophysiological soil for several reasons. Firstly,  
162 neuronal activities propagate within a medium whose number of dimensions is different  
163 from the 3-dimensional “space” of kinematics. A simple centrifugal horizontal motion implies  
164 one single dimension in physical space but as many dimensions as there are parallel  
165 channels transmitting the retinal signals to the abducens and oculomotor nuclei. Secondly,  
166 from the retinal input to the motor output, the pattern of neuronal connectivity (divergence  
167 followed by convergence) suggests multiple homothetic transformations (expansions and  
168 contractions) between the anatomical relays. In other words, the activity which is elicited by  
169 a stimulus, even very small, is not rigid; any gaze or target situation measured here and now  
170 is not reducible to a point of coordinates (x,y,z,t). Thirdly, compared to its changes of  
171 coordinates, a moving target does not yield mirror-like changes of activity in the brain  
172 networks: residual activities persist, as shown in the superior colliculus for example (Goffart  
173 et al., 2017b). The instantaneous velocity of an eye movement is the outcome of action  
174 potentials emitted by motor neurons under the influence of neurons distributed in several  
175 other regions and during a longer time interval. Moreover, correlations between firing rate

176 and velocity can be observed as secondary consequences of changes in alertness or  
177 motivation (e.g., Takikawa et al. 2002). Saccades are indeed slowed and the firing rate of  
178 saccade-related neurons reduced when the alertness declines (e.g., Henn et al., 1984; Fuchs  
179 et al., 1993; Soetedjo et al., 2000). Finally, recordings in the cat have revealed stronger  
180 correlations between the firing rate of motoneurons and the change in muscle force (Davis-  
181 Lopez de Carrizosa et al., 2011). Therefore, the correlation between the firing rate of single  
182 neurons and eye kinematics must be interpreted with the greatest caution, especially for  
183 those neurons located several synapses upstream from the motor neurons.

## 184 **5. TARGET VELOCITY AS A STIMULUS FOR PURSUIT**

185 The suggestion that pursuit consists of matching the eye and target velocities can be  
186 traced back to the studies of Rashbass (1961) and Robinson (1965). It pervades so much the  
187 contemporary sciences of eye movements that most reviews declare visual pursuit as  
188 involving a negative feedback loop reducing a difference between estimates of target and  
189 eye velocities. However, rigorously speaking, when target distance (position error), velocity  
190 or acceleration are said to be stimuli influencing pursuit eye movements, the causal  
191 relationship should be restricted to the sets of numerical values which belong to the same  
192 medium (the physical world) and for which the tools of kinematics have proven their  
193 efficiency.

194 Rashbass (1961) designed an oculomotor task where gaze, instead of shifting toward  
195 a target moving toward the foveal field, drifts away from it, in the same direction as the  
196 target motion but with a lower speed. This observation was taken as evidence that target  
197 velocity is a stimulus for pursuit eye movements. In this task, the target appears at a slightly  
198 eccentric location before moving slowly toward the foveal field. The slow motion must start

199 from an eccentric location whose numerical value is approximately 0.15 to 0.2 times the  
200 target speed. If its speed is less than  $10^\circ/s$ , the target must start from a location which, at  
201 most, is situated 2 degrees from the center of the foveal field. The target *center* is then  
202 located at the edge of the foveal field. However, as explained at the beginning of our  
203 chapter, the fact that the target is a small spot does not imply that its cerebral image is  
204 point-like. A saccade is not launched toward its location because the equilibrium that  
205 characterizes gaze direction is not broken; the visuo-oculomotor saccadic system is within a  
206 mode where opposite commands counter-balance each other (figure 1). We shall now see  
207 that the slow eye movement does not require an encoding of target velocity for its initiation  
208 and maintenance.

## 209 **5. PURSUIT AS SUSTAINED IMBALANCE.**

210 The slow eye movement in the same direction as the target motion (but away from  
211 its physical location) tells us that its generation involves another kind of symmetry breaking.  
212 During horizontal target motions, it results from an imbalance between commands that  
213 tonic neurons in the left and right NPH/MVN exert upon the motor and internuclear neurons  
214 in the abducens nucleus (McFarland and Fuchs, 1992; Scudder et al., 1992). Their bilateral  
215 equilibrium would be broken by any asymmetrical excitation, for instance in the visual input  
216 from the pretectum. The imbalance of activity between the left and right nuclei of the optic  
217 tract (NOT) could also be completed by asymmetric input from the left and right paraflocculi.

218 Unless the drug diffuses toward the pretectum, drifts do not happen during unilateral  
219 inactivation of rostral SC: the monkey is able to maintain stable gaze. Its direction is offset  
220 with respect to the target with an angle which is relatively constant, even while the monkey  
221 pursues a moving target (Hafed et al., 2008). Despite the mismatch between gaze and target

222 directions, the pursuit is preserved. Comparable observations have been shown after caudal  
223 fastigial inactivation (see figures 1 in Burrelly et al., 2018a, 2018b). Made in experimentally-  
224 induced pathological conditions, they indicate that the target does not have to be centered  
225 within the foveal field for being smoothly pursued. Several behavioral experiments in the  
226 normal subject actually demonstrated this possibility (Fuchs, 1967; Pola and Wyatt, 1980;  
227 Robinson, 1965; Segraves and Goldberg 1994; Winterson and Steinman, 1978).

228         Thus, during the Rashbass' task, a velocity signal is not necessary to explain why gaze  
229 moves away from an approaching target. The motion of the target image across the foveae  
230 yields asymmetrical activity between the left and right NOT (Gamlin 2006; Hoffmann et al.  
231 2009; Mustari and Fuchs 1990). Then, the fact that the slow eye movement persists and  
232 increases to reach the same speed as the target, in spite of the diminishing velocity error can  
233 be explained by the maintenance of the same unbalanced drive from the tonic neurons in  
234 NPH/MVN to the motor neurons.

## 235 **6. CONCLUSION.**

236         During the past decades, notions of kinematics were used to “decode” the firing rate  
237 of neurons and to unravel the neurophysiology of tracking eye movements. The  
238 appropriateness of these notions to a medium radically different from the physical world  
239 was not questioned. Instead of embedding the eye movement kinematics in the firing rate of  
240 central neurons, we propose that saccadic and slow eye movements reflect the dynamics of  
241 transitions (from unbalanced states to equilibrium) of bilateral activity carrying directional  
242 tendencies.

243           Thus, the target foveation consists of dynamically adjusting the balance (symmetry)  
244 between opposing tendencies emitted in the left and right parts of the brain, as proposed  
245 for the control of fixation (Goffart et al., 2012; Guerrasio et al., 2010), saccade trajectory  
246 (Bourrelly et al., 2018a; van Gisbergen et al., 1981; Goffart et al., 2003, 2004) and pursuit  
247 (Bourrelly et al., 2018b; Hafed et al., 2008). Concerning the question how eye movements  
248 reach the target speed, the acceleration would involve recruitment: increasing the firing and  
249 number of motion-related neurons moves the eyes faster whereas decreasing them reduces  
250 their velocity. Then, understanding the cerebral control of pursuit eye movement requires  
251 characterizing the adjustment of the appropriate population size through recruiting neurons  
252 and increasing the occurrence of synchronized action potentials at post-synaptic levels  
253 (Goffart et al. 2017a; 2018).

254

## 255   **REFERENCES**

256 Anderson, R.W., Keller, E.L., Gandhi, N.J., Das, S., 1998. Two-dimensional saccade-related  
257 population activity in superior colliculus in monkey. *J. Neurophysiol.* 80, 798-817.

258 Bourrelly, C., Quinet, J., Goffart, L., 2018a. The caudal fastigial nucleus and the steering of  
259 saccades toward a moving visual target. *J. Neurophysiol.* 120, 421-438.

260 Bourrelly, C., Quinet, J., Goffart, L., 2018b. Pursuit disorder and saccade dysmetria after  
261 caudal fastigial inactivation in the monkey. *J. Neurophysiol.* 120, 1640-1654.

262 Davis-Lopez de Carrizosa, M.A., Morado-Díaz, C.J., Miller, J.M., de la Cruz, R.R. and Pastor,  
263 A.M., 2011. Dual encoding of muscle tension and eye position by abducens motoneurons. *J.*  
264 *Neurosci.* 31, 2271-2279.

265 Dias, E.C., Segraves, M.A., 1999. Muscimol-induced inactivation of monkey frontal eye field:  
266 effects on visually and memory-guided saccades. *J. Neurophysiol.* 81, 2191-2214.

267 Dorris, M.C., Munoz, D.P., 1995. A neural correlate for the gap effect on saccadic reaction  
268 times in monkey. *J. Neurophysiol.* 73, 2558-2562.

269 Fuchs, A.F., 1967. Saccadic and smooth pursuit eye movements in the monkey. *J. Physiol.*  
270 191, 609–631.

271 Fuchs, A.F., Robinson, F.R., Straube A., 1993. Role of the caudal fastigial nucleus in saccade  
272 generation. I. Neuronal discharge patterns. *J Neurophysiol.* 70, 1712–1740.

273 Gamlin, P.D., 2006. The pretectum: connections and oculomotor-related roles. *Prog. Brain*  
274 *Res.* 151, 379-405.

275 Goffart, L., Burrelly, C., Quinet, J., 2017a. Synchronizing the tracking eye movements with  
276 the motion of a visual target: basic neural processes. *Prog. Brain Res.* 236, 243-268.

277 Goffart, L., Burrelly, C., Quinton, J-C., 2018. Neurophysiology of visually-guided eye  
278 movements: Critical review and alternative viewpoint. *J. Neurophysiol.* 120, 3234-3245.

279 Goffart, L., Cecala, A., Gandhi, N., 2017b. The superior colliculus and the steering of saccades  
280 toward a moving visual target. *J. Neurophysiol.* 118, 2890–2901, 2017b.

281 Goffart L, Chen LL, Sparks DL., 2003. Saccade dysmetria during functional perturbation of the  
282 caudal fastigial nucleus in the monkey. *Ann. N. Y. Acad. Sci.* 1004, 220-228.

283 Goffart, L., Chen, L.L., Sparks, D.L., 2004. Deficits in saccades and fixation during muscimol  
284 inactivation of the caudal fastigial nucleus in the rhesus monkey. *J. Neurophysiol.* 92, 3351-  
285 3367.

286 Goffart, L., Hafed, Z.M., Krauzlis, R.J., 2012. Visual fixation as equilibrium: evidence from  
287 superior colliculus inactivation. *J. Neurosci.* 32, 10627–10636.

288 Goffart, L., Pélisson, D. 1998. Orienting gaze shifts during muscimol inactivation of caudal  
289 fastigial nucleus in the cat. I. Gaze dysmetria. *J. Neurophysiol.* 79, 1942-1958.

290 Guerrasio, L., Quinet, J., Büttner, U., Goffart, L., 2010. Fastigial oculomotor region and the  
291 control of foveation during fixation. *J. Neurophysiol.* 103, 1988-2001.

292 Hafed, Z.M., Chen, C.Y., Tian, X., 2015. Vision, perception, and attention through the lens of  
293 microsaccades: Mechanisms and implications. *Front. Syst. Neurosci.* 9, 167.

294 Hafed, Z.M., Krauzlis, R.J., 2012. Similarity of superior colliculus involvement in microsaccade  
295 and saccade generation. *J. Neurophysiol.* 107, 1904-1916.

296 Hafed, Z.M., Goffart, L., Krauzlis, R.J., 2008. Superior colliculus inactivation causes stable  
297 offsets in eye position during tracking. *J. Neurosci.* 28, 8124-8137.

298 Henn, V., Baloh, R.W., Hepp K., 1984. The sleep-wake transition in the oculomotor system.  
299 *Exp Brain Res.* 54, 166-176.

300 Hoffmann, K.P., Bremmer, F., Distler, C., 2009. Visual response properties of neurons in  
301 cortical areas MT and MST projecting to the dorsolateral pontine nucleus or the nucleus of  
302 the optic tract in macaque monkeys. *Eur. J. Neurosci.* 29, 411-423.

303 Jürgens, R., Becker, W., Kornhüber, H., 1981. Natural and drug-induced variations of velocity  
304 and duration of human saccadic eye movements: evidence for a control of the neural pulse  
305 generator by local feedback. *Biol. Cybern.* 39, 87-96.

306 Kornhüber, H.H., 1971. Motor functions of cerebellum and basal ganglia: the  
307 cerebellocortical saccadic (ballistic) clock, the cerebellonuclear hold regulator, and the basal  
308 ganglia ramp (voluntary speed smooth movement) generator. *Kybernetik* 8, 157-162.

309 Krauzlis, R.J., Goffart, L., Hafed, Z.M., 2017. Neuronal control of fixation and fixational eye  
310 movements. *Philos. Trans. Roy. Soc. Lond. B Biol. Sci.* 372, 20160205.

311 Lisberger, S.G., Morris, E.J., Tychsen, L., 1987. Visual motion processing and sensory-motor  
312 integration for smooth pursuit eye movements. *Annu. Rev. Neurosci.* 10, 97-129.

313 May, P.J., Hartwich-Young, R., Nelson, J., Sparks, D.L., Porter, J.D., 1990. Cerebellotectal  
314 pathways in the macaque: implications for collicular generation of saccades. *Neuroscience*  
315 36, 305–324.

316 McFarland, J.L., Fuchs, A.F., 1992. Discharge patterns in nucleus prepositus hypoglossi and  
317 adjacent medial vestibular nucleus during horizontal eye movement in behaving macaques.  
318 *J. Neurophysiol.* 68, 319-332.

319 McIlwain, J.T., 1976. Large receptive fields and spatial transformations in the visual system.  
320 *Int. Rev. Physiol.* 10, 223–248.

321 Moschovakis, A.K., Scudder, C.A., Highstein, S.M., 1996. The microscopic anatomy and  
322 physiology of the mammalian saccadic system. *Prog. Neurobiol.* 50, 133-254.

323 Mustari, M.J., Fuchs, A.F., 1990. Discharge patterns of neurons in the pretectal nucleus of  
324 the optic tract (NOT) in the behaving primate. *J. Neurophysiol.* 64, 77-90.

325 Noda, H., Sugita, S., Ikeda, Y., 1990. Afferent and efferent connections of the oculomotor  
326 region of the fastigial nucleus in the macaque monkey. *J. Comp. Neurol.* 302, 330-348.

327 Nowak, L.G., Bullier, J., 1997. The timing of information transfer in the visual system. In  
328 Extrastriate Cortex (eds J. Kaas, et al.) pp 205-241. Plenum Press, Vol. 12.

329 Ohtsuka, K., Sato, H., Noda, H., 1994. Saccadic burst neurons in the fastigial nucleus are not  
330 involved in compensating for orbital nonlinearities. *J. Neurophysiol.* 71, 1976-1980.

331 Optican, L. M., & Quaia, C. 2002. Distributed model of collicular and cerebellar function  
332 during saccades. *Ann. N. Y. Acad. Sci.* 956, 164-177.

333 Otero-Millan, J., Macknik, S.L., Martinez-Conde, S., 2014. Fixational eye movements and  
334 binocular vision. *Front. Integr. Neurosci.* 8, 52.

335 Pola, J., Wyatt, H.J., 1980. Target position and velocity: the stimuli for smooth pursuit eye  
336 movements. *Vision Res.* 20, 523-534.

337 Quinet, J., Goffart, L., 2005. Saccade dysmetria in head-unrestrained gaze shifts after  
338 muscimol inactivation of the caudal fastigial nucleus in the monkey. *J. Neurophysiol.* 93,  
339 2343-2349.

340 Rashbass, C., 1961. The relationship between saccadic and smooth tracking eye movements.  
341 *J. Physiol.* 159, 326-338.

342 Robinson, D.A., 1965. The mechanics of human smooth pursuit eye movement. *J. Physiol.*  
343 180, 569-591.

344 Robinson, D.A., 1975. Oculomotor control signals. In: Lennerstrand G, Bach-y-Rita P (eds)  
345 Basic mechanisms of ocular motility and their clinical implications. Pergamon, Oxford, pp  
346 337-374.

347 Robinson, D.A., 1992. Implications of neural networks for how we think about brain function.  
348 Behav. Brain Sci. 15, 644-655.

349 Robinson, F.R., Straube, A., Fuchs, A.F., 1993. Participation of caudal fastigial nucleus in  
350 saccade generation. II. Effects of muscimol inactivation. J Neurophysiol 70, 1741–1758.

351 Sato, H., Noda, H., 1992. Saccadic dysmetria induced by transient functional decortication of  
352 the cerebellar vermis. Exp. Brain Res. 88, 455-458.

353 Scudder, C.A., Fuchs, A.F., 1992. Physiological and behavioral identification of vestibular  
354 nucleus neurons mediating the horizontal vestibuloocular reflex in trained rhesus monkeys.  
355 J. Neurophysiol. 68, 244-264.

356 Scudder, C.A., Kaneko, C.R.S., Fuchs, A.F., 2002. The brainstem burst generator for saccadic  
357 eye movements: a modern synthesis. Exp. Brain Res. 142, 439-462.

358 Segraves, M.A., Goldberg, M.E., 1994. Effect of stimulus position and velocity upon the  
359 maintenance of smooth pursuit eye velocity. Vision Res. 34, 2477-2482.

360 Soetedjo, R., Kaneko, C.R., Fuchs, A.F., 2000. Evidence that the superior colliculus  
361 participates in the feedback control of saccadic eye movements. J. Neurophysiol. 87, 679-  
362 695.

363 Sommer, M.A., Wurtz, R.H., 1998. Frontal eye field neurons orthodromically activated from  
364 the superior colliculus. J. Neurophysiol. 80, 3331-3335.

365 Sparks, D.L., 2002. The brainstem control of saccadic eye movements. Nat. Rev. Neurosci. 3,  
366 952–964.

367 Sparks, D.L., Holland, R., Guthrie, B.L., 1976. Size and distribution of movement fields in the  
368 monkey superior colliculus. *Brain Res.* 113, 21–34.

369 Takikawa, Y., Kawagoe, R., Itoh, H., Nakahara, H., Hikosaka, O. 2002. Modulation of saccadic  
370 eye movements by predicted reward outcome. *Exp Brain Res.* 142, 284-291.

371 Thier, P., 2011. The oculomotor cerebellum. In: S.P. Liversedge, I. Gilchrist and S. Everling  
372 (Eds). *The Oxford handbook of eye movements.* Oxford University Press.

373 van Gisbergen, J.A.M., Robinson, D.A., Gielen, S. A., 1981. A quantitative analysis of  
374 generation of saccadic eye movements by burst neurons. *J. Neurophysiol.* 45, 417–442.

375 Winterson, B.J., Steinman, R.M., 1978. The effect of luminance on human smooth pursuit of  
376 perifoveal and foveal targets. *Vision Res.* 18, 1165-1172.

## 377 **FIGURE LEGENDS**

378 Figure 1: Visual fixation as equilibrium. A saccade or a slow eye movement is not initiated  
379 when the visuo-oculomotor system is within a mode where opposite commands issued by  
380 the left and right superior colliculi counterbalance each other. For generating saccadic and  
381 pursuit eye movements, the symmetry breaking involves different groups of neurons.

382 Saccades involve neurons located in the pontomedullary reticular formation whereas slow  
383 eye movements involve neurons in the pontine nuclei and the thalamus (see Burrelly et al.,  
384 2018b and Goffart et al., 2018 for more explanations). The bilateral fastigial activity also  
385 contributes to the muscle tone which specifies the horizontal orientation (yaw) of the head  
386 (Goffart and Pélisson, 1998; Quinet & Goffart, 2005).

