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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

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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 $10^\circ/\text{s}$, 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

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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).

