Early head movements elicited by visual stimuli or collicular electrical stimulation in the cat

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Abstract

During the course of previous recordings of visually-triggered gaze shifts in the head-unrestrained cat, we occasionally observed small head movements which preceded the initiation of the saccadic eye/head gaze shift toward a visual target. These early head movements (EHMs) were directed toward the target and occurred with a probability varying between animals from 0.4% to 16.4% (mean = 5.2%, n = 11 animals). The amplitude of EHM ranged from 0.4° to 8.3° (mean = 1.9°), their latency from 66 to 270 ms (median = 133 ms) and the delay from EHM onset to gaze shift onset averaged 183 ± 108 ms (n = 240). Their occurrence did not depend on visual target eccentricity in the studied range (7–35°), but influenced the metrics and dynamics of the ensuing gaze shifts (gain and velocity reduced). We also found in the two tested cats that low intensity microstimulation of the superior colliculus deeper layers elicited a head movement preceding the gaze shift. Altogether, these results suggest that the presentation of a visual target can elicit a head movement without triggering a saccadic eye/head gaze shift. The visuomotor pathways triggering these early head movements can involve the deep superior colliculus. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Shifting the line of sight (=gaze) in space between relevant objects requires the transformation of sensory signals into appropriate motor commands for the eyes, the head and eventually the trunk. Saccadic eye movements, frequently studied in isolation by restraining the head, are by far the best understood component of this gaze orienting behavior (for review see Moschovakis, Scudder, & Highstein, 1996). During the last decade, investigators have studied the neural control of the head movement component and tried to determine how and where within the visuo-motor pathways gaze-related signals are transformed into motor commands for the eye and head plants (for review see Guitton, 1992; Sparks, 1999). It has been shown that neuronal activities at the level of the superior colliculus encode the desired displacement of gaze (eye in space), and not the individual eye and head movements (Munoz, Guitton, & Péligon, 1991; Freedman & Sparks, 1997a). In contrast, the transformation of this collicular gaze-related signal into eye and head premotor commands in the brainstem reticular formation is the subject of controversies (Whittington, Lestienne, & Bizzi, 1984; Guitton, Munoz, & Galiana, 1990; Lefêvre & Galiana, 1992; Paré & Guitton, 1998; Phillips, Ling, Fuchs, Siebold, & Plorde, 1995; Phillips, Ling, & Fuchs, 1999; Sparks, 1999).

Beside this neurophysiological approach, the behavioral approach has focused on the coupling between the eye and head movements contributing to the gaze shift and has shown significant variations between experimental conditions and between animal species (see for review Fuller, 1992). First studies reported in human subjects a nearly simultaneous initiation of eye and head movements (Bartz, 1966; Uemura, Arai, & Shimazaki, 1980; Biguer, Jeannerod, & Prablanc, 1982). However, systematic measurements of the delay between saccade onset and head movement onset (eye–
head delay) have revealed different strategies according to subjects and to experimental conditions (Bizzi, Kalil, & Morasso, 1972; Barnes, 1979; Zangemeister & Stark, 1981; Roll, Bard, & Paillard, 1986; Fuller, 1992; Goossens & van Opstal, 1997). Head movements were shown to be initiated earlier and to contribute more to the gaze shift in ‘head movers’ than in ‘non-head movers’. They were also triggered earlier with respect to the ocular saccade when the target was not sharply defined (auditory target, flashed visual target) or eccentric in the visual field. Examples of markedly uncoupled eye and head movements have been reported under conditions of conflict between two sequential visual targets (Ron, Berthoz, & Gur, 1993) or between a visual target and an auditory distractor (Corneil & Munoz, 1999). Another experimental factor influencing eye–head coupling is the initial eye-in-head and head-on-trunk positions, as shown in humans (Becker & Jürgens, 1992; Volle & Guitton, 1993; Fuller, 1996) and in the monkey (Freedman & Sparks, 1997b). The coordination between the eye and head components of gaze shifts quantitatively differs between animal species mainly because of different ocular- and cephalo-motor ranges. Thus, cat and barn owl head movements significantly contribute to most visually-triggered gaze shifts; in addition both the dynamics and the initiation time of eye and head movements have been shown to be strongly coupled (Guitton et al., 1990; Munoz et al., 1991; Masino & Knudsen, 1993).

Over the course of several past studies (Goffart & Pélisson, 1997, 1998; Goffart, Pélisson, & Guillaume, 1998; Pélisson, Goffart, & Guillaume, 1998), we occasionally noted a small head movement triggered by the visual target presentation and temporally dissociated from the subsequent orienting gaze shift. To understand their functional significance and neurophysiological substrate, we analyzed these early head movements (EHMs) elicited by visual stimulation. Low-intensity electrical stimulation was then applied to the superior colliculus of two animals to similarly elicit head movements preceding gaze shift onset. These data have been presented at the ‘Eye Movements and Vision in the Natural World’ symposium held in Amsterdam (September 2000).

2. Methods

The responses examined for the purpose of this paper have been recorded in 11 animals before any invasive experiment (electrode or canula penetration). The methods for recording saccadic gaze shifts in the head-unrestrained cat have been previously described in detail (Goffart & Pélisson, 1998) and will be recalled only briefly.

2.1. Animal preparation

The animals were prepared for the experiments under general anesthesia (pentobarbital sodium, 30 mg/kg i.p. for induction and 1–3 mg/kg per hour i.v. during surgery) and aseptic conditions following the guidelines from the French Ministry of Agriculture (87/848) and from the European Community (86/609/EEC). Two coils were implanted for the recording of gaze and head positions by the search-coil-in-magnetic-field technique (Robinson, 1963). A U-shaped lightweight plastic piece, permitting the painless restraint of the animal’s head during later experimental phases, and plugs soldered to the coil leads were fixed to the skull with dental cement. Craniotomies and placement of recording chambers over the cerebellum and/or superior colliculus was additionally achieved for the purpose of past studies or, in cats L and O, for the SC stimulation.

2.2. Experimental setups and animal training

After recovery, each cat was placed in a hammock that gently restrained the body, without constraint on natural movements of the head. The hammock was placed inside a 1 m coil frame (CNC Engineering) with the head located at the center of the frame. The visual target was a spoon, subtending 3.5° of visual angle, filled with a food purée and fitted with two infra-red diodes that permitted continuous recording of its position (Urquizar & Pélisson, 1992). In the ‘barrier paradigm’, the cat’s task was to orient its gaze towards a target presented to either side of an opaque screen located in a fronto-parallel plane at a distance of 41 cm. Different screen sizes were used to elicit gaze shifts towards targets located at 7°, 15°, 19°, 27° and 35° from the animal’s body sagittal plane. During conditioning, cats were trained to look, before spoon target presentation, at a white plastic bolt (3° of visual angle) located at the center of the screen. The ambient room light was provided through optical fibers and was interrupted in about 90% of the trials by an electronic shutter (response time = 5 ms) at the beginning of the gaze shift, so that the orienting response was completed in darkness. After 2–3 weeks of training including the period of habituation to the hammock, recording sessions started. Each session consisted of 150–400 trials. Each trial was initiated (data acquisition started for 2 s) when the animal looked roughly in the direction of the fixation stimulus. Then, the food target was presented at the edge of the opaque screen pseudo-randomly to the left or right along the azimuth (80% of the trials) or up or down along the vertical axis (20%). Two animals were also tested in a different paradigm. After several recording sessions in the ‘barrier paradigm’, cat I was tested in the ‘hemi-cylindrical paradigm’, by presenting the food target pseudo-randomly through one of the
holes of a hemi-cylindrical screen centered on the animal’s head at a distance of 41 cm. The holes were situated along the azimuth at cat’s head level, at an eccentricity of 12°, 24°, 36° and 48°. Other aspects of the procedure were identical to those in the ‘barrier paradigm’ (a detailed description of these two ‘food target paradigms’ can be found in our previous papers) and notably, the time of manual target presentation relative to the onset of central fixation varied randomly from trial to trial in a wide range (about 300–2000 ms). The second animal (cat X) was first tested in the ‘hemi-cylindrical paradigm’ and then trained to respond to light emitting diodes (LEDs) in the following ‘LED target paradigm’: after fixation for about 1 s, a central LED was turned off and 200 ms later a peripheral LED was pseudo-randomly presented along the azimuth at 10°, 20°, 30° or 40° from the animal’s body sagittal plane. The LED was selected by a computer to correspond to the position of a hand-held food reward which became visible by turning the room lights back on after each trial. Within 1 week of training, the animal produced consistent visually-triggered orienting movements toward LEDs. In all paradigms (food or LED target), the room lights were turned back on after completion of the trial and the animal was rewarded for orienting to the target within a 0.1–1.5 s latency period.

2.3. Electrical microstimulation of the deeper SC layers

In two cats (L and O), after completion of the recording of the visually-triggered movements, electrical stimulation was applied to the SC deeper layers. A tungsten microelectrode with an impedance of 0.2–1 MΩ (Merrill & Ainsworth, 1972) was lowered in the head-restrained animal and the electrode’s entrance into the SC was detected by recording the characteristic visual responses. Stimulation sites were 1.8 mm deep relative to the SC dorsal surface and their location has been checked after the end of the experimental series by post-mortem histological reconstruction of electrolytic lesions. When the electrode was positioned in a suitable site, the head of the animal was freed and stimulation started. Stimulation consisted in trains of cathodal pulses (0.5 ms duration) delivered by a S88 Grass stimulator and a PSIU6 isolation unit. After the threshold current intensity T was measured (minimum intensity allowing to evoke a gaze shift in more than 75% of trials with 300 ms trains at a pulse frequency of 300 pps), we recorded a series of responses evoked with trains of 300 ms, 2 × T and 300 pps. Then, we reduced either current intensity to 1 × T or frequency to 150 pps and recorded two ‘weak stimulation’ series. Quantitative data reported in this paper concerns the frequency tests. Stimulations were applied while the animal was in a lighted environment and looked at the center of the opaque screen, waiting for the presentation of a peripheral food target which was delayed by 1 s relative to stimulation onset. These stimulation trials were pseudo-randomly mixed with visual trials with a relative proportion of 1/1.

2.4. Data recording and analysis

Search coil signals were linearized and scaled on-line by a computer program, providing four signals proportional to the horizontal and vertical positions of gaze (eye-in-space) and head. The calibration of each coil was performed before implantation and checked in vivo, and if necessary amended by presenting the animal an attractive target at different locations. The overall precision of gaze and head measurement was estimated to be ±0.5° and the spatial resolution was 0.25°. The same program computed on-line signals proportional to horizontal and vertical position of spoon target relative to the animal’s longitudinal body axis (Urquizar & Pélisson, 1992).

Gaze, head and target position signals were sampled on a second PC microcomputer (DataWave software, sampling frequency = 500 Hz), displayed on-line and stored to disk for off-line analysis. Analyses were performed with PC software developed in our laboratory. Gaze and head position signals were digitally filtered (FIR filter, 70 Hz cut-off frequency) and differentiated. The onset and termination of gaze shifts and of head movements were detected based on a velocity threshold (30°/s). The results of this automatic process were checked by displaying each analyzed trial and corrected, when required. Most EHMs were too small to be reliably detected based on velocity criteria and were therefore detected manually by setting cursors at their onset and termination. The time of food target presentation was measured as the time the target position signal exceeded the barrier size. Target, eye, head, and gaze movements parameters were then automatically extracted and further processed by Statistica software (StatSoft). In addition to rejecting anticipatory gaze shifts (latency less than 80 ms: Goffart & Pélisson, 1997), the following criteria were used for EHMs detection: EHM onset lags target onset, gaze is stable during EHM, EHM amplitude exceeds 0.4° and the velocity of EHM decreases before gaze shift onset. The last criteria led us to exclude complex head responses in which the EHM and the ensuing orienting head movement associated with the gaze shift fused together, which rendered the detection of EHM offset ambiguous. Note that this velocity criteria implies an underestimation of the proportion of EHMs. Finally, quantitative analyses were restricted to EHMs with a latency larger than 65 ms (rejecting 11 responses out of 251), which is the shortest visuo-motor delay based on the estimates of afferent and efferent delays of SC motor neurons (Guitton & Munoz, 1991; Munoz et al., 1991).
Fig. 1. Representative examples of EHM in response to the presentation of a visual target (cat L). Position and velocity profiles are shown for the horizontal component of gaze (dotted and solid lines) and head (dotted and broken lines) movements. (A, C) two individual trials with the visual target presented at 35° (A) and 27° (C) showing EHM which are temporally distinct from the orienting gaze and head movements; (B, D) magnified view of horizontal gaze (B) and head (D) movements during selected EHM trials.

3. Results

3.1. Qualitative description of EHM

Since the target was presented horizontally in most trials (see Section 2), our analysis focused on horizontal responses. Fig. 1 illustrates representative examples of EHM recorded in cat L when the target was presented at 35° or 27°. EHM are characterized by the following features: they are initiated shortly after presentation of the visual target, they are associated with a compensatory eye movement which prevents any change in gaze position, and they can be dissociated from the head movement associated with the subsequent gaze shift ('orienting head and gaze movements') since they either completely stop or decelerate before the initiation of the orienting gaze shift. This figure also indicates that EHM are more closely timed to target onset than either the orienting gaze or head movements. Using the above defined criteria to detect EHM, we have examined 8790 responses to targets presented along the azimuth and recorded in 11 different animals. Table 1 lists the number and percentage of EHM encountered. It shows that the frequency of occurrence of EHM

<table>
<thead>
<tr>
<th>Cats</th>
<th>Total no. of trials</th>
<th>No. of EHM trials</th>
<th>EHM rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>573</td>
<td>27</td>
<td>4.7</td>
</tr>
<tr>
<td>D</td>
<td>194</td>
<td>15</td>
<td>7.7</td>
</tr>
<tr>
<td>E</td>
<td>1039</td>
<td>29</td>
<td>2.8</td>
</tr>
<tr>
<td>F</td>
<td>961</td>
<td>79</td>
<td>8.2</td>
</tr>
<tr>
<td>G</td>
<td>1123</td>
<td>5</td>
<td>0.4</td>
</tr>
<tr>
<td>H</td>
<td>1152</td>
<td>7</td>
<td>0.6</td>
</tr>
<tr>
<td>I</td>
<td>1232 (540)</td>
<td>29 (17)</td>
<td>2.3 (3.1)</td>
</tr>
<tr>
<td>K</td>
<td>285</td>
<td>9</td>
<td>3.2</td>
</tr>
<tr>
<td>L</td>
<td>815</td>
<td>133</td>
<td>16.4</td>
</tr>
<tr>
<td>O</td>
<td>387</td>
<td>39</td>
<td>10.1</td>
</tr>
<tr>
<td>X</td>
<td>441 (590)</td>
<td>30 (53)</td>
<td>6.8 (9.0)</td>
</tr>
<tr>
<td>All cats</td>
<td>8790</td>
<td>456</td>
<td>5.2</td>
</tr>
</tbody>
</table>

All cats were tested with a food target either in the ‘barrier paradigm’ (cats B–O) or in the ‘hemi-cylindrical paradigm’ (cat X). Data depicted in parentheses have been additionally collected in the ‘hemi-cylindrical paradigm’ (cat I) and in the ‘LED target paradigm’ (cat X). Quantitative analyses have been performed for the present paper on the data of three cats (bold font).
Fig. 2. Effect of target eccentricity on the rate of occurrence of EHM. Symbols represent different animals and the relationship shown by open squares and solid lines represent mean data computed on the three animals. Only cat L has been tested at an eccentricity of 7°. There is no consistent relationship between EHM probability and target eccentricity (see regression analysis in text).

varies strongly among animals (range = 0.4–16.4%, mean = 5.2%). Note that the animal (cat I) who was tested in both ‘food target paradigms’ showed a nearly equal amount of EHM in the two conditions. In addition, cat X also produced EHM when responding to the presentation of a LED target, with a slightly higher rate than in response to a food target. Thus, EHM produced by the 11 animals when they oriented toward a suddenly presented food target are not specific to this ‘barrier paradigm’.

In the following text, we quantitatively analyze EHM collected in cats F, L and O which showed the highest rates of EHM occurrence. Some EHM with a very short latency could in fact be premature head movements anticipating the target presentation. However, after selection of EHM based on the minimum latency criteria (see Section 2), we found that the vast majority of EHM were in fact driven by the visual target since only three were directed in the wrong direction (1.2%, as compared to 240 movements in the correct direction). Only these correctly directed EHM will be considered in the following analyses.

3.2. Quantitative analysis of EHM

3.2.1. Probability of occurrence

Fig. 2 plots the probability of EHM as a function of target eccentricity. As already mentioned, EHM probability is highest for cat L. In this cat, the probability decreases as target eccentricity increases in the 7–35° range. An opposite trend is observed in cats F and O tested in a 15–35° range of target eccentricity. Overall, a correlation analysis performed across all three cats does not reveal any significant relationship (Pearson correlation coefficient \( r = 0.02, P > 0.05 \)).

3.2.2. Timing, metrics and dynamics

The timing of EHM and gaze shift is illustrated in Fig. 3 in which the onset time of each EHM and subsequent gaze shift are plotted relative to the time of target presentation. Note that some EHM have a very short latency relative to the target presentation, reaching the minimum value of 65 ms determined by our selection criterion (see Section 2). To test whether EHM onset was better timed to target presentation or to gaze shift initiation, we computed the delay from target onset to EHM onset (EHM latency = 133 ± 38 ms) and the pre-gaze delay, as the period separating EHM onset from gaze shift onset ( = 183 ± 108 ms). The variability of the former is significantly smaller than the variability of the latter (variance ratio = 0.12), indicating that EHM are better timed to the onset of visual target presentation than to the onset of the ensuing gaze shift. We further note that EHM latency and pre-gaze delay are affected by the position of the visual target, since both parameters significantly increase as a function of
Fig. 4. EHMs main sequence (data from cats F, L, O pooled together). The relationship between peak velocity and amplitude of the horizontal component of EHMs (filled squares) is compared with that of orienting head movements recorded during the same trials (open circles and linear regression).

Most EHMs are very small, their amplitude ranging from 0.4° (criterion minimum amplitude: see Section 2) to 8.3° (mean = 1.9°). In addition, the horizontal amplitude of EHM is positively correlated with the target eccentricity (Pearson correlation coefficient $r = 0.18$, $P < 0.01$). Most EHMs are very small, their amplitude ranging from 0.4° to 8.3° (mean = 1.9°). In addition, the horizontal amplitude of EHM is positively correlated with the target eccentricity (Pearson correlation coefficient $r = 0.18$, $P < 0.01$).

Fig. 4 compares the dynamics of EHMs to that of ‘orienting head movements’ recorded during the same trials. The main sequence relationships of EHMs (filled squares) and orienting head movements (open circles) are shown superimposed. Although the range of EHM amplitude is restricted, there is a clear linear increase of peak head velocity with increasing head amplitude, with a slope steeper than for orienting head movements. To quantitatively test this velocity difference between the two head movement types, we selected head movements within a comparable amplitude range ($5.20 \pm 1.08^\circ$, $n = 13$ and $5.23 \pm 1.42^\circ$, $n = 10$ for EHMs and orienting head movements, respectively) and applied a Student’s $t$-test for independent samples. We found that the peak velocity of EHMs ($71 \pm 14^\circ/s$) is significantly larger than that of orienting head movements ($46 \pm 10^\circ/s$, $t_{21} = 4.6$, $P < 0.001$). In a separate analysis (not shown), we found that the main sequence of orienting head movements recorded during trials without EHM (control head movements) is not statistically different from that of orienting head movements recorded in EHM trials (Student’s $t$-test, $P > 0.05$). Consequently, the peak velocity of EHMs again are faster ($58 \pm 15^\circ/s$) than that of control head movements ($45 \pm 26^\circ/s$, $t_{51} = 2.1$, $P < 0.05$) for matched amplitude responses (EHMs: $4.04 \pm 0.66^\circ$, $n = 25$; control head movements: $4.08 \pm 0.59^\circ$, $n = 28$).

3.2.3. Interactions with saccadic gaze shifts

To investigate whether the presence of an EHM in some trials can affect the subsequent eye/head gaze shift recorded during the same trials, the amplitude and velocity of these gaze shifts are compared to those of gaze shifts recorded in trials without EHM.

In Fig. 5, the amplitude of the horizontal gaze displacement is plotted as a function of the horizontal retinal error (horizontal distance between target position and gaze initial position) for EHM and non EHM trials. Considering first the movements without EHM (panel A), despite some amplitude variability (recall that the data were collected in several experimental sessions in three cats), there is a strong linear relationship between gaze shift amplitude and horizontal retinal error ($r = 0.99$), with a slope near unity (0.91) and a very small y-intercept (0.01°). Considering now the trials with EHM (panel B), the strong relationship between the two variables is maintained ($r = 0.98$, slope = 0.87 and y-intercept = 0.2°), even though a few strongly inaccurate gaze shifts are observed (see ar-
of EHM and the gain of the subsequent gaze shift. Concerning the effect of EHM latency, two subsets of EHM trials are defined according to the median EHM latency (133 ms): a ‘long latency EHM’ group includes all trials with an EHM latency > 133 ms and a ‘short latency EHM’ group includes all remaining EHM trials (EHM latency < 133 ms). The comparison between these two groups fails to reveal any statistically significant difference in gaze shift gain (Student’s t-test, \(t_{(229)} = 0.7, P > 0.05\); 0.90 ± 0.16, \(n = 111\) versus 0.89 ± 0.14, \(n = 120\) for the ‘long latency EHM’s’ group and the ‘short latency EHM’s’ group, respectively). Concerning the effect of EHM amplitude, two subsets of EHM trials are defined according to the median EHM amplitude (1.6°): a ‘large amplitude EHM’ group includes all trials with an EHM > 1.6° and a ‘small amplitude EHM’ group includes all remaining EHM trials (EHM amplitude < 1.6°). Similarly to the previous analysis, the comparison between these two groups fails to reveal any gaze shift gain difference between the ‘large amplitude EHM’ group (0.88 ± 0.17, \(n = 123\)) and the ‘small amplitude EHM’ group (0.91 ± 0.12, \(n = 108\), Student’s \(t_{(229)} = 1.29, P > 0.05\)).

Thus, the presence of an EHM slightly but significantly interferes with the accuracy of the subsequent orienting gaze shift and this influence does not depend on the latency or on the amplitude of EHM.

Fig. 6A shows the influence of EHM on the dynamics of the subsequent gaze shift. The main sequence relationships of the gaze shifts horizontal component are plotted for those gaze shifts preceded (EHM group) or not by an EHM (non EHM group). A large overlap in these relationships is observed between the two groups. However, the peak gaze velocity for the EHM group is on average markedly reduced with respect to the non EHM group. This difference is confirmed statistically when testing 5° gaze amplitude bins centered on −20° (EHM: −300 ± 47°/s, \(n = 19\), non EHM: −363 ± 77°/s, \(n = 209\); Student’s \(t_{(229)} = 3.51, P < 0.001\)) or on 20° (EHM: 281 ± 62°/s, \(n = 23\), non EHM: 354 ± 73, \(n = 201\); Student’s \(t_{(229)} = 4.61, P < 0.001\)). Fig. 6B plots the horizontal peak velocity versus amplitude relationship of orienting head movements recorded during EHM and non EHM trials. In both cases, the head main sequence relationships are nearly linear and the two data sets largely overlap. The comparison between the two groups for 5° head amplitude bins centered on −20 and 20° (same analysis as described above for gaze dynamics) reveals a statistically significant difference in peak head velocity for rightward head movements (EHM: 142 ± 26°/s, \(n = 29\), non EHM: 161 ± 34°/s, \(n = 235\); Student’s \(t_{(226)} = 2.78, P < 0.01\)) but not for leftward movements (EHM: −147 ± 31°/s, \(n = 27\), non EHM: −155 ± 32, \(n = 231\); Student’s \(t_{(256)} = 1.20, P > 0.05\)). In sum, the presence of an EHM differentially influences the dynamics of the
subsequent gaze shift and head movement, with a marked velocity reduction for the former and a slight reduction for the latter.

3.3. EHMs evoked by SC electrical stimulation

Gaze shifts evoked by electrical stimulation of the deep layers of the superior colliculus were recorded in two cats which were previously tested in the visual target paradigm (cats L and O, see Table 1). As already reported (Péligson, Guitton, & Munoz, 1989; Munoz et al., 1991; Paré, Crommelinck, & Guitton, 1994), suprathreshold electrical microstimulation of the SC in head-unrestrained cats evokes short latency gaze shifts with a strong temporal coupling between eye and head movements onset. However, we find that weaker electrical microstimulation (current intensity less than $2 \times T$ and/or pulse frequency less than 300 pps) can elicit head movements which largely precede gaze shift. In fact, decreasing the stimulation pulse frequency to 150 pps is associated with an increase in the latency of both eye and head movements, but the effect is larger for the eye than for the head, resulting in an increased probability of head leading the gaze (84% at 150 pps versus 70% at 300 pps). Notably, this effect of stimulation frequency differs between the two tested animals (cat L: 100 versus 68%; cat O: 68 versus 73%). Fig. 7 shows representative examples of EHMs evoked by SC electrical stimulation in cat L (stimulation parameters: current intensity 10 $\mu$A = $2 \times T$, pulse frequency 150 pps and train duration 300 ms). The head movement begins shortly after stimulation onset, whereas gaze shift onset occurs later and more variably. A distinctive feature of the majority of the electrically-evoked EHMs (95%) is that their velocity remains somewhat constant or increases slightly until gaze shift initiation, after which a fast acceleration is observed corresponding to the 'orienting head movement'. This contrasts with EHMs in the visual paradigm which quickly decelerate and often completely stop before gaze shift initiation, a difference which may be related to the nature of the stimulus (see Section 4). The magnified view (panels B and D) indicates that electrically-evoked EHMs, as reported above for visually-triggered ones, occur without any detectable gaze movement. These examples also emphasize that the synchronization between stimulation onset and EHM onset is much stronger than that between stimulation onset and the onset of either gaze or head orienting movement.

Fig. 7. Representative examples of EHMs evoked by electrical stimulation of the SC deeper layers (cat L). Position and velocity profiles are shown for the horizontal component of gaze (dotted and solid lines) and head (dotted and broken lines) movements. (A, C) two individual EHMs triggered about 50 ms after stimulation onset and characterized by a slowly increasing velocity. (B, D) magnified view of horizontal gaze (B) and head (D) profiles during selected collicular stimulation trials with EHM. Note that EHMs are more strongly timed to stimulation onset than do gaze shifts. SC stimulation parameters: current intensity 10 $\mu$A, pulse width 0.5 ms, pulse frequency 150 pps and train duration 300 ms.
4. Discussion

In this study, we found that cats tested in a visual target orienting paradigm produced in about 5% of the trials a head movement that starts and either strongly decelerates or completely stops before the coordinated eye/head gaze shift. The large majority of these EHMs (98.8%) were directed toward the target and both their latency and amplitude showed a slight, but significant, increase related to the target eccentricity. In addition, they were better timed to the onset of target presentation than to the onset of the gaze shift. Despite their small amplitude, EHMs had faster dynamics than head movements accompanying gaze shifts. We also showed that low intensity constant stimulation of the SC deeper layers can evoke a head movement which precedes the gaze shift and which slowly and continuously accelerates until gaze shift onset. When compared to these head movements, the typical time course of EHMs elicited in the visual paradigm suggests the involvement of a brisk neural activation. Altogether, these observations suggest that EHMs are generated in response to the presentation of the visual target by some phasic activity in the deep superior colliculus. In the following, we argue that EHMs do not include a significant number of anticipatory responses, compare our results with previous reports of EHM, and discuss the possible neural mechanisms involved in the production of EHM.

4.1. EHMs and anticipatory responses

One could argue that, even with the multiple criterion used to select them (see Section 2), a significant proportion of EHMs are premature head movements anticipating target presentation, especially for those with the shortest latencies. It would have been appropriate to record neck EMG activity to directly address this possibility. Indeed, movements with a latency beyond our 65 ms criteria but associated with a low level neck activity initiated around target presentation time could have been classified as anticipatory and rejected. Unfortunately, the experimental constraints imposed by the various projects from which these data were drawn did not allow us to monitor EMG activity. However, if EHMs are anticipatory, one should observe frequent directional errors, i.e. head movements directed away from the target. Thus, the negligible proportion of misdirected head movements (1.2%) indicates that the large majority of EHMs reported in the present paper are not anticipatory.

4.2. Comparison with previous studies

As stated in Section 1, many past studies have revealed the existence of head movements initiated before the onset of a goal-directed gaze shift and have evaluated the various experimental conditions favoring their occurrence (see Fuller, 1992 for review). However, most of them have dealt exclusively with the head movement occurring conjointly with the gaze shift (called in the present study ‘orienting head movement’). Consequently, the lead time of these head movements relative to gaze shift initiation is limited and rarely exceeds 100 ms (mean lead time less than 50 ms according to Fuller, 1992). Notable exceptions have been reported anecdotally in the cat in a gaze orienting paradigm toward LED targets (Guitton et al., 1990). However, only a few examples were shown, with the head movement characterized by a latency of about 100 ms with a small variability, by a low acceleration, and by a re-acceleration about 20 ms after gaze shift onset. More thorough analyses of head movements initiated a long time before the gaze shift were performed by Ron et al. (1993) and by Corneil and Munoz (1999) in human subjects. Under the double stimulation conditions used by these authors (see Section 1), EHMs dissociated from the ensuing orienting gaze shift were frequently reported and notably EHMs which were directed toward the first target step or toward the distractor location, and preceded gaze shifts directed toward the ultimate visual target. According to Corneil and Munoz (1999), signals derived from the distractor and from the target activate separate neuronal populations at the level of the superior colliculus build-up layer and EHMs occur when the ‘distractor population’ is activated before the ‘target population’ but with an intensity insufficient to trigger a gaze shift.

The present study shows for the first time that EHMs clearly dissociated from the orienting gaze shift can be elicited in the cat in a single visual target paradigm. In this case, the dissociation was not spatial, since EHMs and orienting gaze shifts were directed toward the same goal, but temporal. Indeed, EHM initiation occurred long before the triggering of the gaze shift (183 ms on average) and was much more strongly timed to target onset than to the onset of the ensuing gaze shift. We want to mention that EHM and head movement associated with the ensuing gaze shift can sometime coalesce, as seen by some biphasic velocity profiles with an initial low velocity followed by a sharp head re-acceleration around the time of gaze shift onset. However, these ambiguous responses were not included in our sample (see Section 2). The small probability of EHMs analyzed in this paper is thus not surprising given the severity of the selection criteria. In addition, cat eye and head movements are usually strongly coupled and the very existence of EHM in this animal species was unexpected. Despite their paucity and their reduced amplitude, EHMs were found to have slight detrimental effects on the accuracy and velocity of the ensuing gaze shift, which were both slightly but significantly reduced relative to gaze shifts recorded in trials without
EHM. In addition, the reduction of gaze shift amplitude did not depend on the latency or amplitude of EHM. This suggests that the decrement in gaze shift performance cannot be accounted for by a possible increase of gaze latency associated with the occurrence of EHM, or by the slight head deviation which follows EHM, but is more specifically related to the occurrence of the EHM. One may speculate that the production of an EHM is associated with some leakage in neural signals encoding both the metrics and dynamics of the impending gaze shift. Unit recording experiments are necessary to test this hypothesis but in any case, the functional relevance of EHM, if any, is totally uncertain. However, analyzing the factors favoring their occurrence can tell us about their neurophysiological substrate and about the structure of the gaze shift system.

Since the probability of EHM occurrence varied between animals in a 40-fold range, one such factor is related to the experimental subject. Such inter-subject difference can be related to the variable propensity to use the head in orienting gaze, as clearly illustrated in human subjects (Fuller, 1992). The present study further suggests that this propensity can in part be related to the excitability of the tecto-fugal pathways involved in the initiation of head movements without triggering a gaze shift. Indeed, although inferences from only two animals may be anecdotal, we found that weak electrical microstimulations in the SC evoked head movements preceding gaze shifts with a larger probability (100% versus 68%) in the subject who was more prone to produce EHM in the visual paradigm. The occurrence of EHM may also depend on experimental factors such as the saliency of the target which has been shown to affect eye/head coupling in gaze orienting tasks (see for review Fuller, 1992). To test this possibility, we used in two animals a modified food target paradigm and a LED target with a 200 ms gap paradigm, respectively, in addition to the basic paradigm in which a food target appears at the edge of an opaque barrier. The results indicate that the number of EHMs did not strongly depend on the experimental paradigm. We only note that using a LED instead of a piece of food as a visual target led to slightly more frequent EHM in cat X. Further experiments are necessary to confirm this difference and to test whether the temporal gap used in the LED task or the motivational value attached to the target could be responsible for it.

4.3. Neural basis of EHMs

The presence of EHM without concurrent gaze displacement indicates that some neuronal activity generated in response to the visual target presentation can drive the head motor centers without activating the brainstem saccadic burst generator. In addition, since short latency head movements preceding gaze shift onset can be produced by low intensity electrical stimulation of the SC deeper layers, the origin of EHM could be a brief activation of collicular neurons resulting from the visual target presentation. It is known that the SC drives eye and head premotor neurons located in the pontine and medullary reticular formation. Some of these neurons are specifically involved in the generation of saccadic eye movements and are gated off by the OPNs during periods of fixation (Sparks & Mays, 1990; Moschovakis et al., 1996) whereas other neurons receiving inputs from the SC (reticulo-spinal neurons) are involved in the production of both eye and head movements or of head movement alone and are not gated by OPNs (Grantyn, Berthoz, Hardy, & Gourdon, 1992; Robinson, Phillips, & Fuchs, 1994). In addition, at least in the cat, a substantial fraction of collicular output neurons project directly to the spinal cord where they contact the premotor neurons driving head movements (Grantyn & Grantyn, 1982; May & Porter, 1992; Olivier, Chat, & Grantyn, 1991). Thus, through these direct and indirect (reticular) pathways which by-pass the brainstem burst generator for saccades, the SC can selectively drive spinal neurons involved in the generation of head movement while the OPNs inhibit the oculomotor circuitry.

This scheme has been previously proposed by Corneil and Munoz (1999) to account for their data recorded in human subjects. A prediction of their scheme was that electrical stimulation of SC deep layers should be able to trigger head movements alone when a sub-threshold intensity regarding the production of saccadic eye movements is used. Responses reported in the present paper are consistent with this prediction, since low intensity stimulation trains can evoke a head movement more than 100 ms in advance of the saccade onset. These responses are reminiscent of the EHMs observed in the visual target paradigm except for their velocity profile. Indeed most electrically-evoked EHMs show a constantly increasing velocity until the gaze shift was initiated, after which the high head acceleration associated with gaze shift initiation was observed. This can be related to the constant electrical stimulation of the SC. In contrast, visually-triggered EHMs decelerate or even completely stop before gaze shift initiation, implying the involvement of a short lasting neural drive. These data suggest that the collicular activity involved in the production of visually-triggered EHM is a transient activity in the deep collicular neurons resulting from the target presentation. Note that a short lasting activity in the SC deep layers, either a visual burst or a fused visual and motor burst, has been shown to evoke short latency saccadic eye movements in monkeys (Edelman & Keller, 1996; Dorris, Paré, & Munoz, 1997; Sparks, Rohrer, & Zhang, 2000). The fact that EHM is not associated with a concurrent gaze displacement in our
study could indicate that the corresponding SC activity is weak. However, contrary to this hypothesis, we have shown that EHM s have dynamics even slightly faster than regular orienting head movements and have latencies down to the shortest visuo-motor delay (about 65 ms, see Guittot & Munoz, 1991, and Munoz et al., 1991). Thus an alternative explanation of the lack of simultaneous movement of gaze is that, at the time the visual stimulation leads to an intense but sudden activation of the SC deep layers, the saccadic pulse generator is still highly inhibited by OPNs. This would prevent the transient SC activation from triggering a saccadic gaze shift but not from driving the head motor system through the by-pass pathways mentioned above.

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