Typical exogenous covert shift of attention without the ability to plan eye movements

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The automatic allocation of attention to a salient stimulus in the visual periphery (e.g., a traffic light turning red) while maintaining fixation elsewhere (e.g., on the car ahead) is referred to as *exogenous covert shift of attention* (ECSA). An influential explanation is that ECSA results from the programming of a saccadic eye movement toward the stimulus of interest [1, 2], although the actual movement may be withheld if needed. In this paper, however, we report evidence of ECSA in the paralyzed axis of three individuals with either horizontal or vertical congenital gaze paralysis, including for stimuli appearing at locations that cannot be foveated through head movements. This demonstrates that ECSA does not require programming either eye or head movements and calls for a re-examination of the oculomotor account.

To test the oculomotor account, we assessed vertical and horizontal ECSA in three individuals with congenital vertical (VGP) or horizontal (HGP1 and HGP2) gaze paralysis due to hypoplasia of the lower brainstem and 15 typically-developed control participants using a classical target detection task with nonpredictive peripheral cues (See Figure 1A and Supplemental material Experiment 1 for details). Efficient saccadic programming is not innate but requires some learning by trials, errors, and adaptation to develop [3]. Congenital paralysis prevents this development. Accordingly, the oculomotor account predicts that neither VGP, HGP1 or HGP2 should show ECSA in their paralyzed axis [1, 2].

As a group, typically-developed participants showed a faster detection of targets preceded by a valid than by an invalid cue at SOA of 100 ms (i.e., the “cueing effect”) in both axes (horizontal: $t(14)=5.036$, $p<.001$; 23 ± 17 ms; d=1.3; vertical: $t(14)=6.249$, $p<.001$; 23 ± 14 ms; d=1.61; Figure 1B). This cueing effect was transient as it decreased (horizontal: $t(14)=2.092$, $p=.055$; vertical: $t(14)=3.185$, $p=.007$) and became

non-significant (horizontal: $t(14)=2.021$, $p=.063$; 8 ± 15 ms; d=.52; vertical: $t(14)=1.974$, $p=.068$; 8 ± 17 ms; d=.51; Figure 1B) at SOA of 300 ms.

At odds with the prediction of the oculomotor account, HGP1, HPG2 and VGP all showed significant indicators of ECSA in their paralyzed orientation (see Figure 1B and Supplemental results for details). HGP1 showed significant horizontal cueing at 100 ms ($p=.007$) and inhibition of return at 300 ms ($p<.001$). The horizontal version of the task was administered twice to HGP2; both sessions revealed significant cueing at 100 ms ($p=.016$ and $p=.021$) that decreased at 300 ms ($p=.072$ and $p=.301$). VGP's vertical cueing was significant at 300 ms ($p=.006$; $p=.238$ at 100ms). In addition, besides a significantly stronger reversal of the cueing effect in the horizontal axis at 300 ms in HGP1 (modified *t*-test, $p=.01$), all effects were comparable to those found in the controls (see Figure 1, all modified *t* tests for case-control comparisons *ps* > .05) and none of these effects differed more between the paralyzed and non-paralyzed orientation in HGP1, HGP2 and VGP than between the two axes in the control participants (all Bayesian Standardized Difference Tests *Ps* >.1).

To sum up, we report three individuals with congenital gaze paralysis who all showed significant indicators of ECSA in their paralyzed orientation. Although VGP's cueing emerged slightly later (between 100 and 300 ms), three arguments indicate that this delayed effect reflects the normal inter-individual variability in the time course of cueing effects in this task rather than a consequence of her paralysis: (1) this effect was also found in two typically-developed participants; (2) it did not differ significantly from VGP's profile in the non-paralyzed orientation and (3) more importantly, the profiles of HGP1 and HGP2 demonstrates that congenital paralysis per se does not cause a delay in ECSA. Previous studies that failed to find significant signs of ECSA in individuals with gaze paralysis were so far taken as evidence in favor of the oculomotor account

[4, 5]. These conclusions were based on associations of deficits and null results, which are both difficult to interpret. The inferential value of the dissociation between absent oculomotor programming and consistent statistically significant ECSA reported herein supersedes that of these previous findings and force the conclusion that ECSA does not require the programming of eye movements toward the stimulus of interest.

As individuals with gaze paralysis shift their gaze by operating saccadic-like head movements [6], a possibility is that typical ECSA in absence of eye movements could be supported by the programming of head movements. In this case, our results would have to be reinterpreted as evidence about the range of computational and neural plasticity that is possible within the system that underlies ECSA. However, this possibility is challenged by the finding that saccadic head movements are significantly slower than ocular saccades (see Supplemental Experiment 2). Indeed, according to the oculomotor theories [1, 2, 5], factors that affect the speed of saccades should also affect ECSA. To directly test this possibility, we blocked HGP2's head in maximal rotation to the left (about 70°) and compared ECSA for targets that would be reachable or not by the eyes through head rotation (see Figure 1C and Supplemental Experiment 3). If the programming of head saccades was responsible of the cueing effects in Experiment 1, these effects should vanish when the targets are displayed outside the head rotation range. At odds with this possibility, we observed typical and similar cueing effects for targets located within and outside the head rotation range (all *p*s < .02; see Figure 1D and detail in Supplemental Material). This shows that ECSA does not require the ability to program either eye or head movements towards the locus of attention.

Previous studies have shown that the attentional network is equipped with visuo-motor neurons responding to both eye movements and ECSA [9], and some studies reported

that typically-developed participants do not always benefit from exogeneous cues presented outside their oculomotor range [7, 8, but see 10]. Together with our finding, this suggests that ECSA does not result from but may be influenced by oculomotor processes. As such, our findings encourage a shift in the focus of future research toward the fundamental question of how these two distinct but complementary systems influence each other.

Figure Caption:

Figure 1. Target detection tasks. **[A, B]** Experiment 1. **[A]** Time course of a trial. Each trial begins with the presentation of one central and two peripheral white boxes (size: 2.5° x 2.5°) presented at a fixed eccentricity (6°) along either the horizontal or vertical axes (in separated blocks). The central box contains a dot that participants are instructed to fixate during the whole trial. After 1000 ms, the width of the outline of one of the two peripheral boxes increases, providing an “exogeneous cue”. Then, in 80% of the trials a target dot appears in one of the two peripheral boxes either 100 or 300 ms after the onset of the exogeneous cue; the location of the target is not predicted by the position of the cue. In the remaining 20% of the trials, no target appears. Participants have to press a response button as fast as possible whenever a target dot appears in one of the peripheral boxes. The task comprised 10 blocks of 60 trials. **[B]**. Results. Cueing effect (invalid – valid) for Horizontal and Vertical axis as a function of the SOA (100 vs. 300 ms). Positive values represent faster response latencies for valid trials than for invalid ones. Blue (VGP), Green (HGP1) and Orange (HGP2; Square Mark: First session with horizontal axis only; Lines: Second session with horizontal and vertical axes) represent the individual cueing effect of the participants with congenital gaze paralysis. Gray lines represent individual cueing effect of typically developed control participants. Black lines represent the mean cueing effects of the typically

developed control sample. **[C, D]**. Experiment 3. **[C]** Illustration of the experimental set-up. **[D]** Results. Cueing effect of HGP2 for targets that were Reachable or Unreachable by the fovea as a function of SOA (100 vs. 300 ms). Dashed lines represent the individual cueing effect for targets that were Reachable and solid lines for targets that were Unreachable.

Declaration of Interest:

The authors declare no competing interest.

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