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Does more imply better vision?

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Correctly identifying the orientation of objects in space is crucial in many aspects of our life, from daily activities requiring efficient tool use to geometrical learning.

Vannuscorps et al. (2021), in the current issue, finely characterize the interesting case of a woman, Davida, who fails to recognize orientation. Davida, a teenager with a normal medical history, no evidence of cortical abnormalities, and proficient in many visual tasks, performs poorly when she has to process visually the orientation of high contrast 2D shapes. Davida, however, performs perfectly when she is asked to recognize items haptically. The authors interpreted this pattern of deficits as the consequence of an impairment at an intermediate stage of processing from a pure retinotopic frame of reference to a complete spatiotopic representation of space that is independent of our body/gaze direction. The authors refer to this intermediate level as an "intermediate shape-centred representation" (ISCRs) that should correspond to an allocentric frame of reference (centred on the visual object). A large body of literature demonstrates that the brain simultaneously uses many frames of reference (Cohen & Andersen, 2002; Goodale, 2014), and the fact that only one frame can be affected in an otherwise typical woman strongly corroborates the idea of multiple representations, showing the complexity and the fragility of the mechanisms which generate space representation.

Davida's impairments are consistent with damage at an early stage along the cortical visual pathways in contrast to earlier studies that demonstrated damage at a higher tier of multimodal cortical processing (Castaldi et al., 2018; Goodale, 2014). We have recently described a group of children with lesions at the level of the periventricular white matter (PVL) that presented agnosia for mirror orientation that was specific only for diagonal orientations (Castaldi et al., 2018). They consistently confused diagonal orientations with their mirror counterpart, while perception of cardinal orientations was preserved. The deficit generalized to stimuli varying in many low-level features, such as oriented gratings or bars or second order stimuli (i.e., collinear short lines embedded within randomly oriented lines) and also affected the haptic modality. The selectivity of their deficit for diagonal orientations allowed us to test the specificity of this deficit for retinotopic vs. spatiotopic frame of reference and we demonstrated that the visual orientation deficit remained selective for the diagonal orientations in space, indicating that the effect occurred in spatiotopic and not retinotopic coordinates and that it was independent of gravity and of the monitor frame of reference (allocentric representation). One of implications of the deficits described in our PVL patients is that perception of visual orientation of oblique and cardinal stimulus relies on partially separate mechanisms. In this respect it would be interesting to test Davida also with oblique orientations in those conditions that yield high performance with cardinal orientations (e.g., low contrast).

Perception of object orientation in space is achieved through a cascade of visual processing steps, the first of which consists of the identification

of 2D edges and lines allowing for detection and segregation of the objects' borders from the background (Burr et al., 1989, 1992; Morrone & Burr, 1988). There is evidence that the identification and discrimination of edges and lines is supported by regions along both the ventral (V4) and the dorsal stream (caudal part of the intraparietal sulcus, transversal occipital sulcus, dorsal LO and V3A), irrespective of these features being defined by luminance or chromatic contrast (Castaldi et al., 2013; Perna et al., 2005, 2008). Areas along the dorsal pathway presumably start from this local information and compute objects' orientation independently of retinotopic coordinates and even irrespective of the modality, visual or tactile (Kitada et al., 2006), to support action guidance (Goodale, 2014). In the light of this evidence, it is likely that the deficit observed in our PVL patients arises from a lesion of the posterior parietal cortex, which is often reported to be damaged in this pathology (Fiori et al., 2015) and is consistent with their deficit being invariant to spatiotopic coordinates.

The case study of Davida, clearly different from our PVL children, probably occurred at an earlier stage of visual analysis, her dependence on contrast of the orientation deficit strongly supporting this view. She showed small deficits or none at all when the oriented stimuli were blurred or defined by small dots, or lines, or had low contrast (i.e., with low energy stimuli). To interpret these findings, the authors suggested the formation along the extrastriate pathways of multiple independent "intermediate shape-centred representations" (ISCRs) associated with the different visual cues (like colour, luminance, low contrast, high contrast, etc).

However, simpler explanations are equally possible. High contrast stimuli do not always lead to better perception and Davida is not the only patient showing consistent deficits only at high contrast. Tadin's work (2003, 2015) has shown a significant drop in performance for high contrast motion stimuli and we demonstrated it in typical participants as well as those with dyslexia (Bhat et al., 2018; Perani et al., 2021). In Perani et al. (2021) discrimination performance topped between 10% and 30% Michelson contrast and then, surprisingly, dropped at higher contrast, in particular in a subgroup of dyslexic individuals carrying a specific genetic variant (the DCDC2 gene deletion). Interestingly, motion contrast sensitivity and high contrast accuracy in these individuals correlated with the fractional anisotropy in the optic radiations and ventral tracts located in the inferior temporal cortex (inferior longitudinal and inferior fronto-occipital fasciculi), which provide input/output to V1. These deficits are stronger at high spatial frequency stimuli (see Fig 1G&H in Perani et al., 2021), and this is consistent with Davida's perception showing stronger deficits for objects defined by sharp edges. Also, the apparently normal perception that Davida displays in Experiment 5.9, 5.10 and 6.10 is consistent with the hypothesis that high contrast explains the deficit: despite the high contrast of the single dots or pixels, the low density of dots or pixels defining either the shapes or the motion in those paradigms reduce dramatically the effective contrast of the stimuli after accounting for the small blurring associated with normal acuity.

One possible explanation for these and Davida's contrast-dependent deficits might be that contrast is correctly computed in V1, but then the read-out of this first stage by higher order areas (such as V4 in the case of Davida) is impaired. Neural responses to increasing luminance contrast levels are typically described by sigmoid-like functions, defined by a monotonic increase in neural firing with increasing contrast up to a maximum saturation level (Albrecht & Hamilton, 1982). However, this pattern is not common to all neurons in all visual processing stages. For example, neurons in macaque area V4 can display bandpass tuning response for contrast (Sani et al., 2013) during an orientation discrimination task. Interestingly the non-monotonic U-shaped selectivity emerged only later in the neuronal response (at around 150 ms after stimulus onset) suggesting a longer integration time. Sani et al. (2013) proposed that contrast should be considered an independent and basic feature, such as color or orientation, and that the slower non-monotonic type of response might correspond to a contrast categorization stage. Psychophysical studies seem to support the existence of contrast selective neurons in humans as well, by showing that adapting to a contrast defined (2nd order) pattern of fixed contrast impairs the visibility of test patterns composed of elements with straddling contrast (Wolfson & Graham, 2007, 2009). These are hallmarks of a sensory domain which represents contrast flexibly.

In the light of these results, it would be interesting to test Davida with a similar contrast adaptation paradigm to evaluate whether some of these channels

coding for medium to high contrast are impaired and cause the reported orientation deficit.

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