



## OPEN Motor imagery in individuals with congenital aphantasia

Robert Kwaśniak<sup>1✉</sup>, Dariusz Zapala<sup>2</sup>, Paweł Augustynowicz<sup>2</sup> & Magdalena Szubielska<sup>2</sup>

Individuals who experience aphantasia have an inability to create sensory mental images, what lead to a range of cognitive and behavioral differences compared to the general population. However, little is known about how this phenomenon affects the creation of motor imagery. Our study aims to check the differences in changes of hemoglobine concentration between individuals with congenital aphantasia (AG) and control group (CG) during creating a kinesthetic (KMI) or visual-motor (VMI) representation of movement. Twenty participants (10 AG) who participated in the experiment were matched by age, gender, education level, and handedness. During data collection, a hemodynamic signal was recorded using functional near-infrared spectroscopy (fNIRS). The participants performed a procedure that enabled the control of perspective and cognitive strategies during motor imagery using a haptic interface. The results indicate that AG demonstrate reduced oxygenated hemoglobin concentration in the right middle frontal gyrus and right motor cortex regions. The findings suggest that AG primarily rely on semantic or kinesthetic strategies, while CG tend to use visual cognitive strategies during both the KMI and VMI tasks. Furthermore, we propose that AG may exhibit difficulty with the process of reorienting attention from exogenous to endogenous control.

**Keywords** Aphantasia, Motor imagery, fNIRS, Haptic interface

Aphantasia is a phenomenon that refers to the inability to create sensory mental images. While most of the individuals experience aphantasia from birth<sup>1</sup>, there is a group of people who acquired it as a result of neurological injury or psychiatric disorders<sup>2</sup>. Previous research suggests distinguishing between aphantasia involving a single sensory modality (e.g., visual) or multiple sensory modalities (e.g., visual and auditory)<sup>3</sup>. Accordingly, aphantasia is treated as a heterogeneous phenomenon characterized by distinct subgroups with different patterns of difficulties in creation of sensory imagery<sup>4</sup>.

The most commonly used method to identify individuals with aphantasia is the Vividness of Visual Imagery Questionnaire (VVIQ)<sup>5</sup>. However, despite its prevalence as a self-reporting method, this technique has certain inherent limitations, including a potential susceptibility to the respondent's level of metacognition and a low resistance to response bias<sup>6</sup>. Furthermore, researchers do not use the questionnaire consistently, which presents an additional challenge in the reliable and accurate identification of individuals with aphantasia<sup>7</sup>. Another method for identifying individuals with aphantasia is the “binocular paradigm”. In a study conducted by Pearson and Keogh<sup>8</sup>, the researchers presented the participants with a cue in the form of the letter “R” or “G” to signify Gabor patches of different colors. Subsequently, the participants were requested to visualize the presented stimulus. In the next step, the participants were presented with the two stimuli, each to a distinct eye. The participants exhibited a tendency to perceive one of the two stimuli, rather than alternately switching between them in consciousness. Participants rated the image's vividness on a scale of 1 to 4. Then they selected which Gabor patch had been seen more often. The researchers demonstrated that the priming effect in the binocular paradigm was reduced in individuals with aphantasia compared to the control group. This indicates that attempting to imagine a previously observed Gabor patch did not result in its perception during binocular rivalry in individuals with aphantasia. This effect was subsequently evidenced in studies<sup>9,10</sup>.

The latest method is based on the pupilographic index, which was investigated by Kay et al.<sup>11</sup>. Pupil diameter occurs in response to psychophysiological regulation of light access to the retina<sup>12</sup>. In response to darkness, the pupil dilates, whereas in response to brightness, it constricts. This mechanism, known as the pupil response index (PRI), is characterized by distinct neural pathways responsible for regulating the dilation and constriction of the pupil in response to different stimuli<sup>13</sup>. However, the pupil not only responds involuntarily to the perception of visual stimuli<sup>14</sup> or affective emotional states<sup>15</sup>, but also to higher-order cognitive activity<sup>16</sup>, such as mental imagery. The researchers demonstrated that individuals with aphantasia exhibited no pupillary

<sup>1</sup>Institute of Philosophy, The John Paul II Catholic University of Lublin, Lublin 20-950, Poland. <sup>2</sup>Department of Experimental Psychology, Institute of Psychology, The John Paul II Catholic University of Lublin, Lublin 20-950, Poland. ✉email: robert.michal.kwasniak@gmail.com

response compared to baseline to luminance during imaginary task. However, they did demonstrate perceptual responsiveness and dilation to luminance and cognitive load<sup>11</sup>.

Identifying individuals with aphantasia allows for further investigation into the differences in mental processes such as memory, planning and their neuronal basis resulting from the absence of sensory, mental images. Keogh et al.<sup>1</sup> indicated that individuals with aphantasia did not exhibit differences in visual working memory capacity or spatial working memory capacity compared to a control group. However, significant differences were observed in the strategies adopted by those with aphantasia in all the memory tasks conducted. Other studies also showed no significant differences in visual working memory performance between individuals with aphantasia and controls<sup>17,18</sup>. A further study examined the neural underpinnings of visual imagery vividness through the use of resting-state functional magnetic resonance imaging (fMRI)<sup>19</sup>. The study included both aphantasia and hyperphantasics (individuals who report their visual imagery as vivid as the images they perceive). The findings revealed that individuals with hyperphantasia exhibited stronger functional connections between frontal areas and the visual cortex than those with aphantasia.

One type of imagery is motor imagery (MI), which can be defined as the mental simulation of a movement, without actually performing it<sup>20</sup>. It can be divided into visual-motor imagery (VMI) and kinesthetic motor imagery (KMI). Visual-motor imagery, in turn, is divided into internal (IVMI) and external imagery (EVM). Internal visual-motor imagery involves imagining a representation of movement from a first-person perspective. In contrast, external visual-motor imagery involves creating a representation of one's own or another's movement from a third-person perspective. Kinesthetic motor imagery (KMI), on the other hand, consists of bodily sensations<sup>21</sup>. To date, only one study has examined the process of motor imagery and motor system activation during action observation between aphantasia individuals and controls<sup>22</sup>. The researchers used TMS to measure corticospinal excitability, as well as to obtain a subjective report on the vividness of the imagery and the extent to which it is used in everyday life. All of the participants were instructed to imagine a maximal pinch movement in both the visual and kinaesthetic modalities and to observe a video showing a pinch movement. The results demonstrated that the amplitude of motor-evoked potentials (MEPs) increased for the control group during kinesthetic motor imagery and action observation relative to rest, but not during visual motor imagery. Conversely, no such increase in MEP was observed in any of the conditions for the group of aphantasia.

In conclusion, previous research has applied a range of techniques to identify individuals with aphantasia (AG) and subsequently examine their cognitive processes through the use of behavioral experiments or techniques such as fMRI and TMS. It appears that AG exhibits reduced connectivity between the frontal and visual cortex<sup>19</sup>. Additionally, they may adopt alternative strategies such as kinesthetic or semantic when performing tasks as a compensatory mechanism for the inability to create sensory mental images. To date, it is still uncertain how AG declaratively reports reduced vividness of visual imagery creates MI, which is inherently multisensory. Nevertheless, the studies on AG lack several crucial elements that this study aims to address.

The first problem arises from the fact that previous research has not separated MI. The act of imagining movement can be performed from a variety of perspectives, including first-person (1PP) and third-person (3PP). The failure to take these strategies into account may result in a blurring of the observed effects, due to the different neural underpinnings of these two perspectives<sup>23,24</sup>. In addition, the participant may employ various strategies, including visual-motor or kinesthetic. Given that AG utilizes different cognitive strategies, it is essential to construct the experimental procedure in a way that allows for the control of the imagined movement. The role of higher cognitive functions, such as working memory and attention, in the creation of motor imagery for AG in comparison to a CG also remains unknown.

The methodology employed was adapted from the procedure described by Zapala et al.<sup>25</sup>, in which the impact of brief visual deprivation on cerebral oscillations was investigated using electroencephalography (EEG) and haptic interface. In this experiment, we used functional near-infrared spectroscopy (fNIRS) due to its greater spatial resolution and higher resistance to motion artifacts compared to EEG<sup>26,27</sup>. Only relative changes in oxygenated hemoglobin (HbO) were analyzed, as it is more sensitive to changes in blood flow (CBF) and it has higher signal-to-noise ratio (SNR) than HbR<sup>28</sup>. Additionally, muscle tension effects on motor performance were controlled through the use of electromyography (EMG). This was done to prevent the introduction of false results due to uncontrolled hand movements during the imaging of movement. Finally, we adapted the pupil measurement experiment of Kay et al.<sup>11</sup> to allow for control of this variable between AG and CG under perceptual and imaginary conditions.

Furthermore, we hypothesize that the increase in oxygenated hemoglobin (HbO) in the left motor cortex area will be higher in the KMI condition for AG compared to the CG. Kinesthetic imagery consists of representations that evoke motor simulations of one's own body<sup>29</sup>. These simulations can take an abstract form. The findings of the study conducted by<sup>30</sup> indicate that early visual representations are vulnerable to perceptual interference, whereas areas of the parietal cortex demonstrate resilience to this phenomenon. This increases the likelihood that the minds of individuals, who are unable to generate sensory mental images, store these images in the parietal cortex in an abstract form [after 26]. Because of that a more embodied effect in AG could be observed, supposing that the mechanism of mental imagery and unconscious sensory simulations happening offline is separable.

We postulated that a greater increase of HbO in the right middle frontal gyrus would be in CG in the KMI condition compared to AG. Activation of the right middle frontal gyrus is associated with reorienting attention from exogenous to endogenous attentional control<sup>31</sup>. The presented task, which uses kinesthetic imagery, requires the participant to adopt an egocentric perspective. Adopting this approach for AG in relation to greater embodiment, is a more accessible strategy that does not necessitate as much cognitive effort as the CG. Additionally, we hypothesized that AG, in comparison to CG, will demonstrate increased activation of HbO in the left middle frontal gyrus region during VMI task. Activation of the left middle frontal gyrus has been linked to semantic processing<sup>32,33</sup>. It appears that AG, who are unable to engage in sensory mental imagery, may be able

to compensate for this deficit through the use of semantic strategies, whereby auditory and linguistic cues are integrated.

Furthermore, additional exploratory analyses were conducted on L-MFG/R-MFG lateralization and LMC/RMC lateralization in the KMI and VMI conditions. This was done to further test the hypothesis that the groups could differ in the asymmetry of the activation of observed effects<sup>34,35</sup>.

The last hypothesis concerns the stronger activation of HbO in visual areas in the VMI condition in CG compared to AG. Given that AG are incapable of creating sensory mental images in their minds, it can be postulated that the activity of these areas will be weaker in this group. One study measured the activity of the primary visual cortex in AG, demonstrating that the neuronal response in these individuals during a perceptual task was ipsilateral. Furthermore, it was found that neural activation evoked by perception was weaker in AG compared to CG<sup>36</sup>.

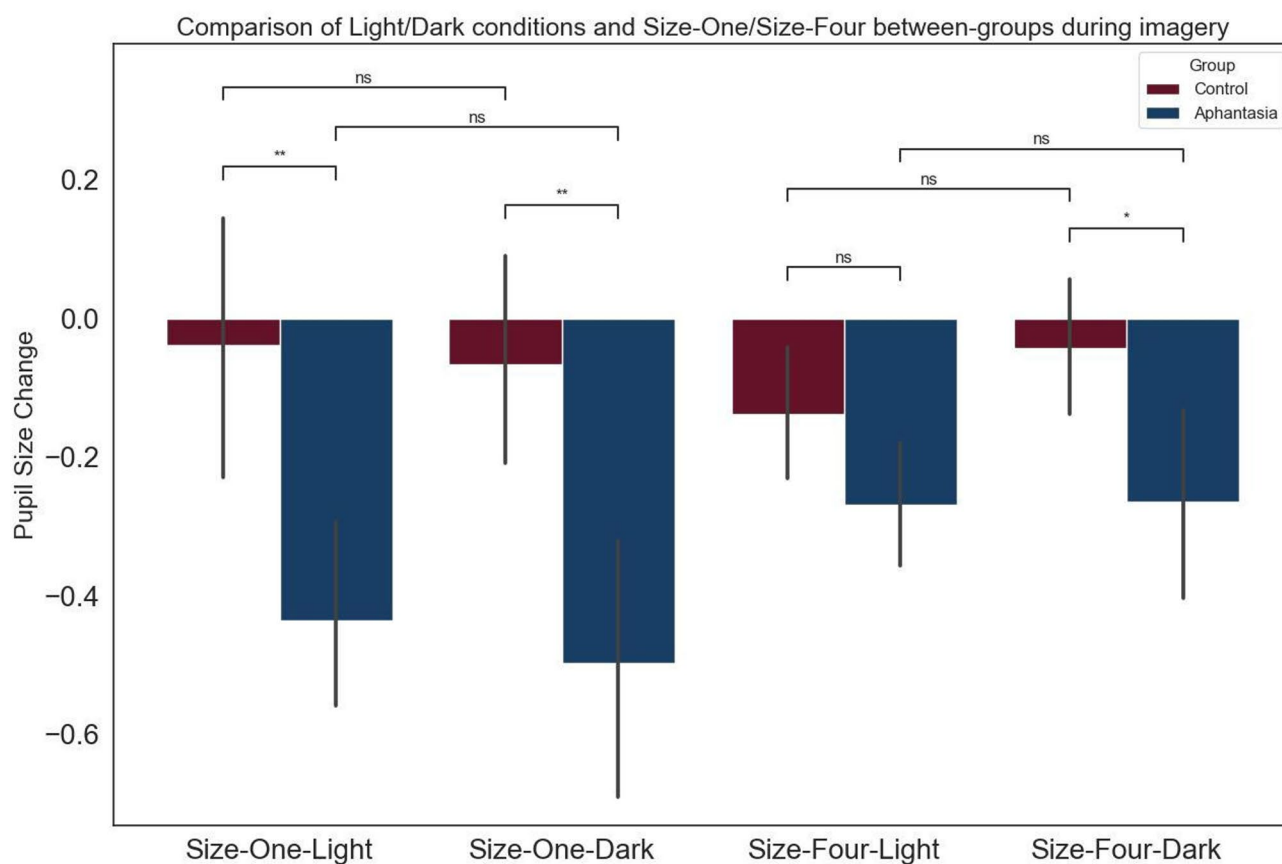
## Results

### Pupil size analysis

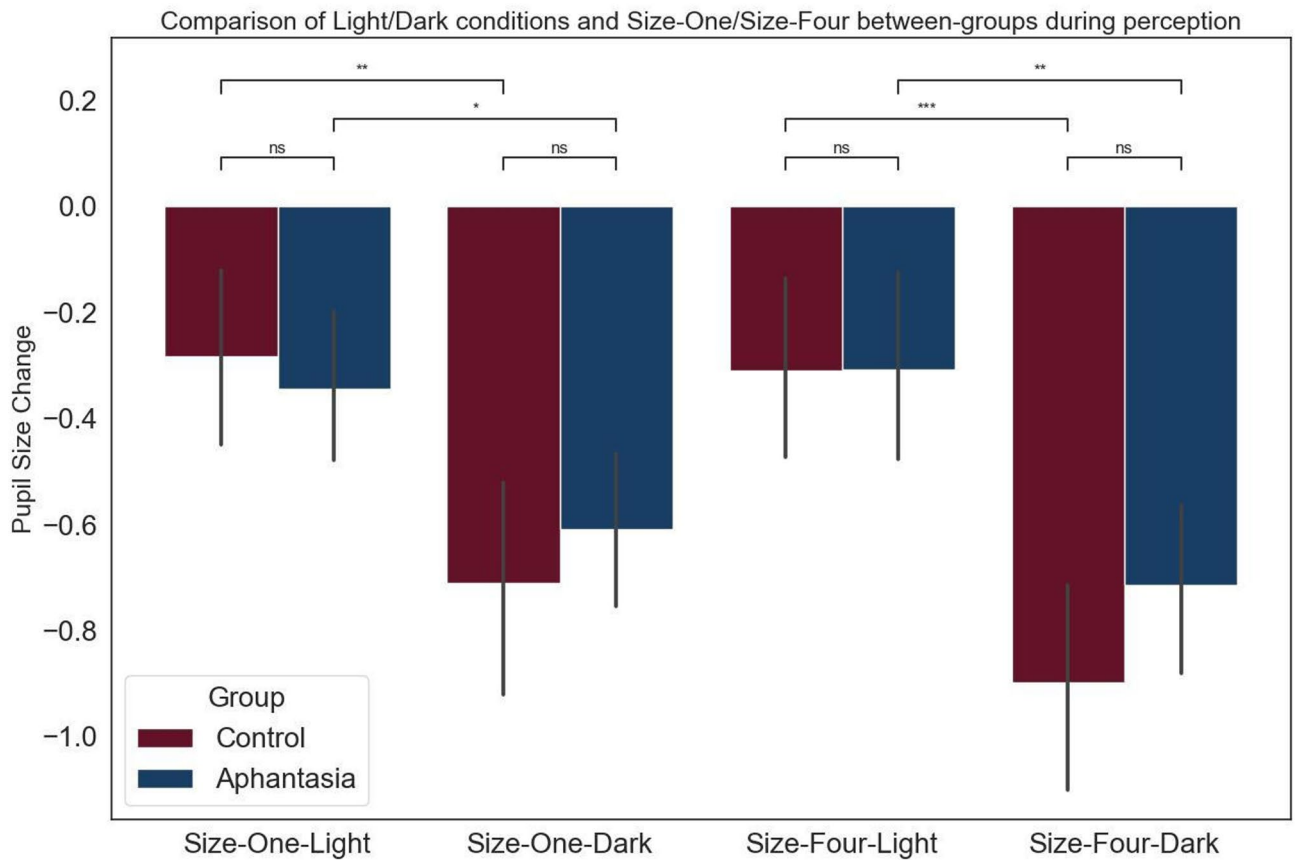
To test whether the pupil measure would distinguish between the AG and CG groups, the following analyses were conducted.

To control pupil size we performed repeated measures ANOVA with a between-subject factor GROUP (Aphantasia/Control) and within-subject SIZE (One/Four), LUMINANCE (Light/Dark), covariate as an age. Dependent value was a mean difference score between imagery and baseline (mm). To test the assumption of equality of variances, a Levene's test was performed and the conditions were met. There was a main effect of the GROUP  $F(1,17) = 14.632$ ;  $p = 0.001$ ;  $\eta^2 = 0.463$  (Fig. 1). AG have a more strongly negative difference score ( $M = -0.367$  mm;  $SE = 0.055$  mm) than CG ( $M = -0.072$  mm;  $SE = 0.055$  mm). Interaction between SIZE\*GROUP were also found  $F(1,17) = 4.929$ ;  $p = 0.040$ ;  $\eta^2 = 0.225$ . The Bonferroni post-hoc test ( $p < 0.05$ ) showed a difference for the AG, between size-one ( $M = -0.467$  mm;  $SE = 0.078$  mm) and size-four ( $M = -0.267$  mm;  $SE = 0.052$  mm).

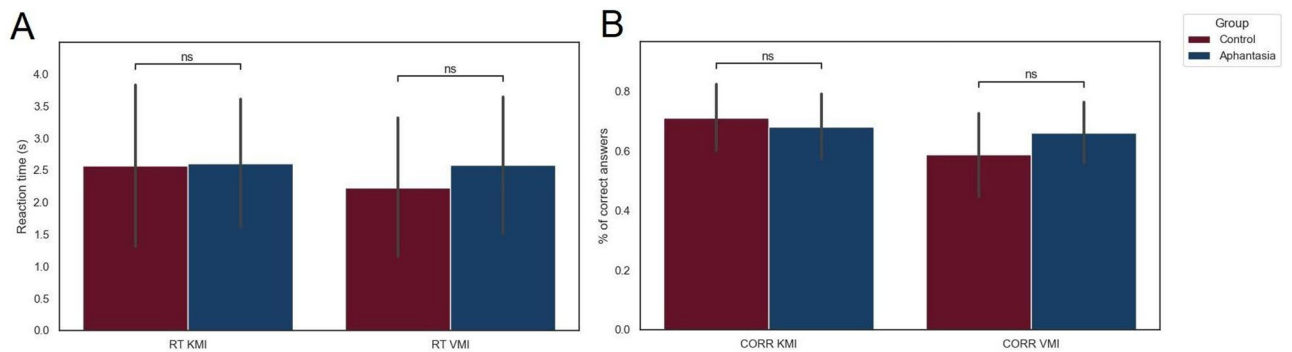
Next we assessed, if there is any significant difference in mean pupil size to perception stimuli. Again we performed repeated measures ANOVA with a between-subject factor GROUP (Aphantasia/Control) and within-subject SIZE (One/Four), LUMINANCE (Light/Dark) with covariate as an age. There was no main effect of a GROUP  $F(1,17) = 0.225$ ;  $p = 0.641$  and no significant interactions between groups (Fig. 2). We have observed a significant main effect of LUMINANCE ( $F(1,17) = 4.965$ ;  $p = 0.040$ ;  $\eta^2 = 0.226$ ). The Bonferroni post-



**Fig. 1.** Comparison of changes in pupil size from baseline (mm) during imagining between the CG and AG in the light/dark and one/four conditions.



**Fig. 2.** Comparison of changes in pupil size from baseline (mm) during perception between the CG and AG in the light/dark luminance and one/four size conditions.



**Fig. 3.** Behavioral response. **(A)** Comparison of reaction time (ms) between aphantasia group and control group for KMI and VMI. **(B)** Comparison of response correctness (%) between the aphantasia group and the control group for KMI and VMI.

hoc test ( $p < 0.001$ ) showed stronger negative mean value of pupil-size during perception for dark stimuli ( $M = -0.733$  mm;  $SE = 0.66$  mm), than for light stimuli ( $M = -0.311$  mm;  $SE = 0.062$  mm).

**Behavioral performance**

First, it was tested whether there were differences between the control and experimental groups in the accuracy (coefficient from 0 to 1) on both the KMI and VMI tasks. An independent samples t-test showed no significant differences between the groups in the correctness of the results for both KMI,  $t(18) = 0.596$ ;  $p = 0.558$  and VMI (Fig. 3B),  $t(18) = -1.299$ ;  $p = 0.210$ .

It was also tested whether there were differences between the control and experimental groups in reaction times (seconds) in the KMI and VMI task. A t-test showed no significant differences between groups in reaction time for KMI,  $t(18) = -0.070$ ;  $p = 0.945$  and for VMI (Fig. 3A),  $t(18) = -0.677$ ;  $p = 0.507$ .

### EMG activity

An analysis of variance with repeated measures was performed to test whether the groups differed in terms of muscle tone in the KMI and VMI tasks during imagery or grip squeeze with the right hand. No main effect of GROUP was observed,  $F(1,18) = 0.043$ ;  $p = 0.839$ . However, we have noted significant main effect of CONDITION  $F(1,18) = 170.651$ ;  $p < 0.001$ ;  $\eta^2 = 0.253$ ; TASK  $F(1,18) = 69.085$ ;  $p < 0.001$ ;  $\eta^2 = 0.159$  and the interaction between CONDITION\*TASK  $F(1,18) = 54.169$ ;  $p < 0.001$ ;  $\eta^2 = 0.117$ . The post-hoc comparisons with Bonferroni correction show significant difference between KMI ( $M = 10.314$ ;  $SE = 1.583$ ) (Fig. 4A) and VMI conditions (Fig. 4B) ( $M = 7.803$ ;  $SE = 1.324$ ) during imagery  $t(1,18) = 3.133$ ;  $p = 0.034$ .

### fNIRS

#### Left middle frontal gyrus

A repeated ANOVA was conducted with a between-subject factor GROUP (Aphantasia/Control) and within-subject factors IMAGERY (KMI/VMI) and CHANNEL (S9D7, S10D7, S10D9). There were no significant main effects of the GROUP  $F(1,15) = 0.214$ ;  $p = 0.650$ , IMAGERY  $F(1,15) = 0.144$ ;  $p = 0.709$  and CHANNEL  $F(2,32) = 0.767$ ;  $p = 0.473$ .

#### Right middle frontal gyrus

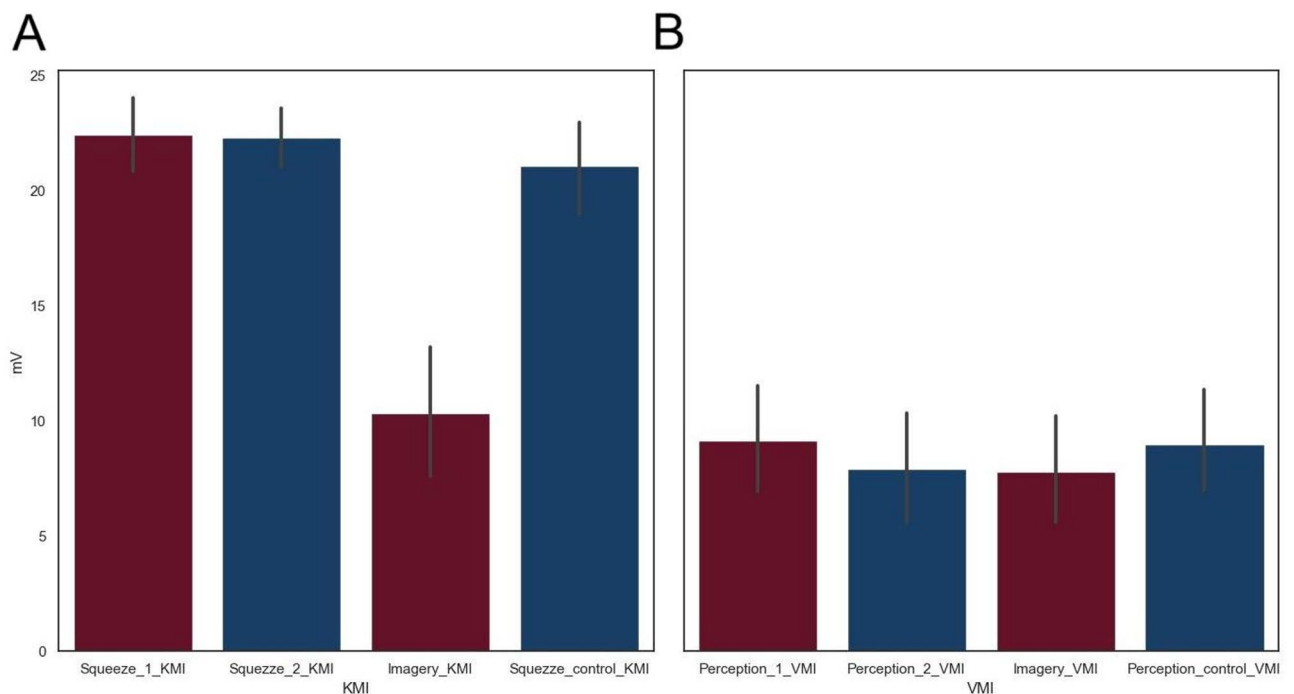
A repeated ANOVA was conducted with a between-subject factor GROUP (Aphantasia/Control) and within-subject factors IMAGERY (KMI/VMI) and CHANNEL (S11D9, S11D8, S9D8). The main effect of the GROUP was significant  $F(1,12) = 5.537$ ;  $p = 0.037$ ,  $\eta^2 = 0.316$  (Fig. 5). The post-hoc tests with Bonferroni correction showed that the control group presented stronger activation of HbO in R-MFG ( $M = 0.047 \mu\text{M}$ ;  $SE = 0.012 \mu\text{M}$ ), than the aphantasia ( $M = 0.009 \mu\text{M}$ ;  $SE = 0.010 \mu\text{M}$ ) group. There were no significant main effects of the IMAGERY  $F(1, 12) = 1.260$ ;  $p = 0.284$  and CHANNEL  $F(2, 24) = 2.081$ ;  $p = 0.147$ .

#### Left motor cortex

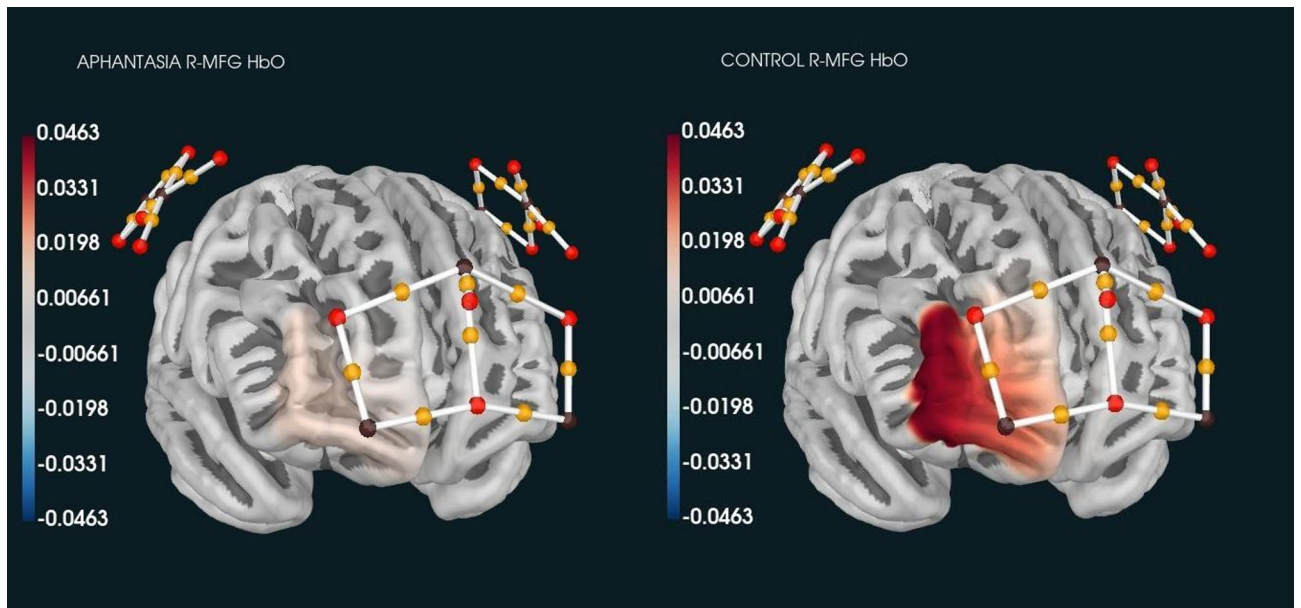
A repeated ANOVA was conducted with a between-subject factor GROUP (Aphantasia/Control) and within-subject factors IMAGERY (KMI/VMI) and CHANNEL (S1D1, S3D1, S5D1, S5D3, S7D1, S7D3). There were no significant main effects of the GROUP  $F(1,8) = 1.155$ ;  $p = 0.314$ , IMAGERY  $F(1, 8) = 0.297$ ;  $p = 0.601$ ; CHANNEL  $F(5, 40) = 1.378$ ;  $p = 0.253$ .

#### Right motor cortex

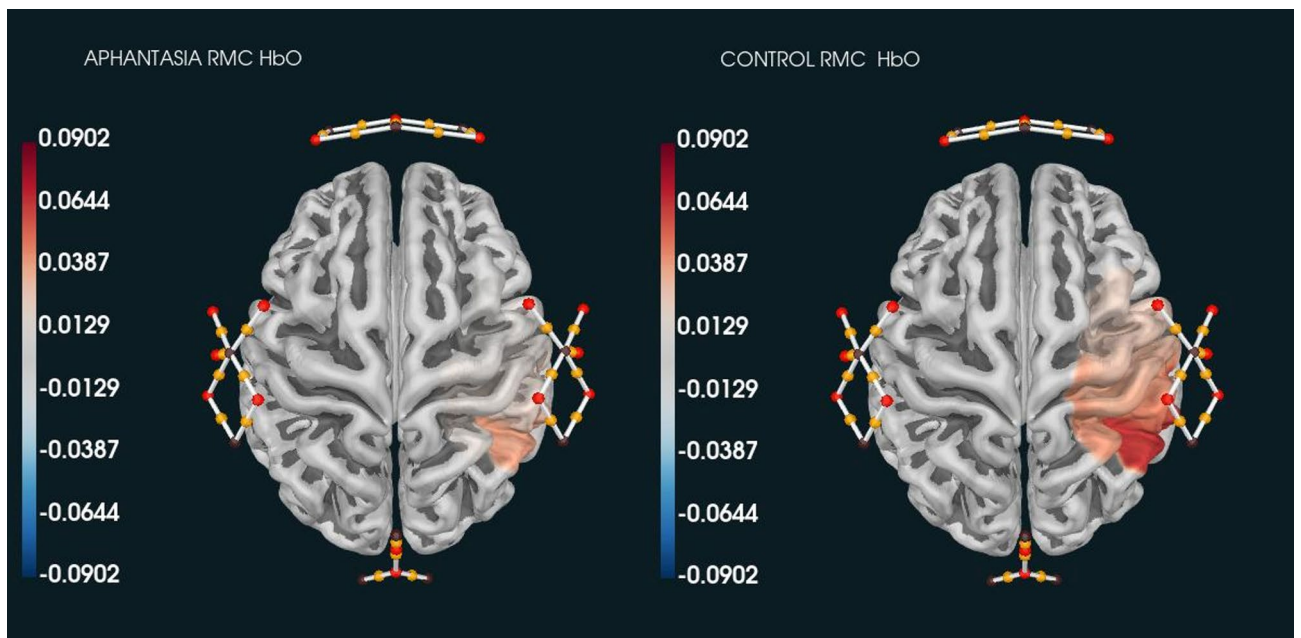
A repeated ANOVA was conducted with a between-subject factor GROUP (Aphantasia/Control) and within-subject factors IMAGERY (KMI/VMI) and CHANNEL (S2D2, S4D2, S6D2, S6D4, S8D2, S8D4). The main



**Fig. 4.** EMG activity. **(A)** Comparison of the squeeze of the haptic interface handle and imaginary for the KMI condition. **(B)** Comparison of perception visual motor imagery task and imagery for the VMI condition.



**Fig. 5.** Comparison of relative changes in oxygenated hemoglobin concentration ( $\mu\text{M}$ ) for R-MFG area (S11D9, S11D8, S9D8) between aphantasia and control group.



**Fig. 6.** Comparison of relative changes in oxygenated hemoglobin concentration for RMC area (S2D2, S4D2, S6D2, S6D4, S8D2, S8D4) between aphantasia group and control group.

effect of the GROUP was significant  $F(1, 9) = 7.834$ ;  $p = 0.021$ ;  $\eta^2 = 0.465$  (Fig. 6). The control group showed higher activation of HbO in RMC ( $M = 0.061 \mu\text{M}$ ;  $SE = 0.009 \mu\text{M}$ ) than Aphantasia ( $M = 0.022 \mu\text{M}$ ;  $SE = 0.010 \mu\text{M}$ ). Mauchly's sphericity test was violated for the CHANNEL within-subject factor  $W(1, 14) = 0.017$ ;  $p = 0.014$ ;  $e = 0.763$ . Using the Huyn-Fieldt correction for sphericity, a statistically significant result was obtained  $F(5, 34.357) = 3.134$ ;  $p = 0.028$ . The post-hoc test with Bonferroni correction showed higher activation of HbO in S8D4 ( $M = 0.050 \mu\text{M}$ ;  $SE = 0.010 \mu\text{M}$ ) channel than S8D2 channel ( $M = 0.016 \mu\text{M}$ ;  $SE = 0.007 \mu\text{M}$ ).

#### Brain activity lateralization

Paired t-test was performed between HbO averages for averaged channels separately for two ROIs (middle frontal gyrus and motor cortex) including L-MFG (S9D7, S10D7, S10D9) and R-MFG (S11D9, S11D8, S9D8) and

LMC (S1D1, S3D1, S5D1, S5D3, S7D1, S7D3) and RMC (S2D2, S4D2, S6D2, S6D4, S8D2, S8D4) to determine lateralized responses per condition (KMI/VMI) separately for each group of individuals with aphantasia and controls.

The paired t-test revealed a statistically significant effect ( $t(9) = 2.370$ ;  $p = 0.042$ ;  $d = 0.22$ ) between L-MFG ( $M = 0.016$ ;  $SD = 0.040$ ) and R-MFG ( $M = -0.000$ ;  $SD = 0.036$ ) in the aphantasia group when considering the KMI condition. Activation of L-MFG was stronger than R-MFG. No significant effects were observed for the VMI condition in the aphantasia group or for the control groups in the KMI and VMI conditions.

The results of the paired t-tests did not yield any statistically significant effects between the LMC and RMC in either the individuals with aphantasia or in the control group, regardless of the experimental condition (KMI or VMI).

## Discussion

The primary purpose of this study was to determine differences in changes of HbO concentration between AG and CG during the creation of kinesthetic (KMI) and visuo-motor (VMI) representations of movement. In the motor imagery task, the HbO changes in four regions of the left and right middle frontal gyrus, as well as the left and right motor cortex, were compared between two groups that consisted of 10 AG and the 10 CG. The present study did not confirm all hypotheses but firstly it revealed significant differences between the groups in the activation of the right middle frontal gyrus (R-MFG). AG demonstrated a smaller increase in HbO in R-MFG in comparison to the CG. The R-MFG would be expected to control the reorientation from exogenous to endogenous attention<sup>31</sup>. Endogenous attentional control is associated with top-down processes, whereas exogenous control is linked to bottom-up processes. The dorsal attention network (DAN) underlies endogenous attentional control<sup>37</sup> and the ventral attention network (VAN) (Chica et al., 2013) underlies exogenous control. The DAN is thought to control stimulus-response selection, while the VAN is responsible for sending reorientation signals to the dorsal network to interrupt ongoing processing and direct attention to the exogenous stimulus<sup>38,39</sup>.

The Pearson et al.<sup>40</sup> perspective is based on the assumption that there is both ventral and dorsal visual imagery, which are separate from each other. In AG, it is the dorsal pathway that is impaired and the ventral pathway that is working. This explains why AG retains spatial imagery but cannot create sensory mental images. According to our results we therefore suggest that AG do not so much have a damaged dorsal network resulting in a lack of volitional visual imagery, but that AG may have a problem with switching between endogenous and exogenous attentional control. Difficulties in attentional switching would include the task of KMI and VMI, which, despite their distinct nature, evoke the same first-person perspective. However, research to date has mainly focused on the endogenous component of attention and has not touched on the mechanism of reorientation in AG<sup>9</sup>.

We also showed that AG exhibit weaker HbO activation in the right motor cortex (RMC) area than CG. Kinesthetic imagery elicits greater activation in sensorimotor regions contralateral to the movement performed<sup>41</sup>. In contrast, visual-motor imagery evokes a less lateralized response due to its visual component in sensorimotor regions, which in turn induces stronger activation in areas of visual cortex<sup>42,43</sup>. Given the observed effect in our study, there is evidence to suggest that CG may have employed a cognitive strategy priming effect for the both tasks. In the case of kinesthetic imagery, it can be postulated that the participants in this group may have employed a visual strategy, which is attributable to a number of factors. Previous research has demonstrated that closing and opening the eyes can modify activation patterns in both the KMI and VMI tasks within this paradigm<sup>25</sup>. Additionally, access to visual stimuli may result in the use of a visual strategy due to its greater accessibility. Secondly, the participants were not divided into groups based on whether they would perform the KMI or VMI procedure. Such a procedure would assist in determining the potential impact of integrating the strategies employed during these tasks. Therefore, the observed difference on the ipsilateral side between AG and the CG for the movement performed may be indicative of either a more pronounced difference in activation relative to the left motor cortex for AG, or the use of semantic or kinesthetic perspective in both a KMI and VMI by AG. Accordingly, we conducted an additional examination of the lateralization of the four selected areas of interest (LMC, RMC, L-MFG, R-MFG) in each of the two groups. The observed significant left lateralization of the middle frontal gyrus (MFG) in the KMI task in AG lends support to the second interpretation of the result. This stronger activation of the L-MFG may be related to semantic and linguistic processing<sup>44,45</sup>.

The absence of observed behavioral differences between the groups further explains the utilization of kinesthetic perspective in visual tasks by AG. It is intriguing to note that AG, who lack the capacity for sensory mental imagery, demonstrated comparable performance on the VMI task to those with visual imagery. This raises the question of the extent to which the tasks performed by the participants engage their imaginative processes, as opposed to, for instance, memory processes, whereby the participant recalls an earlier imagination rather than actively creating it anew. Furthermore, it is important to consider that AG reported deficits in both internal and external visual motor imagery at the declarative level, as indicated by their responses on the MIQ-3 questionnaire. Furthermore, AG did not indicate a reduction in self-report kinesthetic imagery with MIQ-3 questionnaire compared to the control group. This raises the question of how AG in our sample were able to perform equally on the task as the CG, despite the fact that it forced the use of visual component imagery. Such a possibility of using non-visual and sensorimotor strategies by AG was observed in visual working memory tasks, where the participants similarly used their strategies as effectively as in our study<sup>46</sup>.

In order to explain the relationship between the declarative difference in the MIQ-3 questionnaire and VMI/KMI scores at the behavioral level, it is necessary to consider the following elements. The participants are required to indicate on the MIQ-3 questionnaire the ease or difficulty with which they perceive or feel a given stimulus. Despite having the scale as a reference point for their feelings or perceptions, adequately locating them requires not only insight into one's own bodily or imaginary experience, but also an adequate focus of consciousness at the phenomenal level. It is essential that the participant be adequately trained to ensure the accuracy and reliability of the results. In addition, the participant point of reference is his internal feelings, taken

as an internal criterion. In contrast, the motor imagery task required participants to align their responses with an external criterion, namely the comparison of imagined resistance or visual-motor stimuli. The distinction in the evaluation criteria indicates that the two methods quantify motor imagery in disparate ways. Consequently, individuals may exhibit variability in their level of evaluation or insight into their own motor imagery, which may not be evident when comparing their motor imagery with an external criterion.

The analysis of changes in pupil dilation indicated that both AG and CG demonstrated pupil contraction in response to perceptual brightness and dilation with effort. Moreover, AG exhibited a more pronounced pupil contraction in response to imagery than CG. However, when comparing the results for the imagery condition, a statistically significant interaction between size and group was observed only for AG. Additionally, no main effect of luminance was observed for the CG in imagery condition. The results partially confirm that the paradigm can help to identify AG even in smaller samples of participants ( $n = 10$ ).

Some of the limitations of these results should be presented. It should be noted that the Questionnaire on Mental Imagery (QMI)<sup>47</sup>, which could have been used to identify potential subtypes of aphantasia<sup>4</sup> was not included in the participant recruitment process. Furthermore, the participants were not asked to describe verbally the cognitive strategies they employed in motor tasks. Including a qualitative component in future studies, such as an in-depth or microphenomenological interview, would be beneficial to gain a more comprehensive understanding of the cognitive processes involved. In addition, we were forced to not include the visual cortex area in the analyses due to too many rejected channels. A further challenge is that the analyses were conducted using a relatively small number of channels in the fNIRS, which may restrict the ability to interpret the observed results.

## Conclusion

The findings of our study provide insight into the KMI and VMI of AG and CG. In accordance with our hypothesis, AG may exhibit a preference for the use of semantic or kinesthetic strategies in a KMI task. Furthermore, these individuals also demonstrate the usage of these strategies in a VMI task, whereas CG in both the VMI and KMI tasks seem to rely on visual strategies. We propose an interpretation of this result based on behavioral and hemodynamic data. Moreover, we find the pupil measure from the Kay et al. experiment<sup>11</sup> could be a valuable objective tool for identifying group of AG. Our results also raise questions about the mechanism of endogenous and exogenous attentional control AG as a potential source of the inability to create sensory mental images. However, due to the limitations of the current research, further investigation is required to address these issues in future.

## Methods

### Participants

Participants were recruited through online advertisement on social media. In addition, we used the “snowball method”, asking the recruited AG to ask if anyone close to them also voluntarily does not experience mental images. To test the hypotheses of IMAGERY \* GROUP a priori interaction, we conducted power analyses using Glimpse 3.1.3 software<sup>48</sup>. A statistical test with Greenhouse-Geisser correction was used due to the correction for non-sphericity. We assumed equality of groups (1:1) and  $SD = 0.048$  for our dependent variable of relative changes in oxygenated hemoglobin concentration based on other studies using fNIRS in the motor imagery area<sup>49</sup>. The results indicate a minimum sample size of  $N = 18$  to observe the IMAGERY \* GROUP interaction (test power = 0.825,  $\alpha = 0.05$  two-tailed, LEAR correlation matrix). Out of a group of 26 qualified individuals (13 AG; 13 CG matched), 20 individuals (10 AG; 10 CG matched) decided to participate in the study.

20 right-handed participants aged 18–46 years ( $M = 26.1$ ;  $SD = 9.85$ ) participated in the study. There were 10 participants in AG (age 19–46 years;  $M = 26.2$ ;  $SD = 9.86$ ) and the CG (age 19–46 years;  $M = 26$ ;  $SD = 10.37$ ). The AG group was matched with CG participants in terms of gender, age, education and handedness. All of the individuals performed the Edinburgh Handedness Inventory (EHI), which assesses hand dominance<sup>50</sup>, the polish version of the Movement Imagery Questionnaire (MIQ-3)<sup>51</sup> which allows to determine the level of imagery ability and second polish version of Vividness of Visual Imagery Questionnaire (VVIQ-2PL) which assesses the vividness of visual imagery<sup>52</sup>. Two people were rejected from the MIQ-3 questionnaire due to incomplete responses. Furthermore, the fact that individuals with a history of neurological injury or medication were excluded at the recruitment level suggests that the recruited individuals were characterized by congenital, not acquired, aphantasia. These assumptions were validated through the process of asking participants during the recruitment phase.

Written informed consent to participate was obtained from all participants who participated in the experiment. AG were matched to the CG for age, education and handedness. All of the participants declared beforehand that they were not taking any psychiatric medication and had no history of neurological injury. At the end of the whole experiment, all of the participants were paid 100 PLN (around 23 euro). The study was conducted in compliance with the Declaration of Helsinki approved by the Ethics Committee of the Institute of Psychology at the John Paul II Catholic University of Lublin (NR: KEBN 28/2024 IP KUL, date 03.06.2024).

### Questionnaire assesment

First, it was tested whether the groups differed in terms of lateralisation as measured by LQ score in the EHI. The Mann-Whitney U-test showed no significant differences,  $U = 42$ ;  $p = 0.533$  for the CG ( $Me = 100$ ;  $SD = 9.478$ ) and the AG ( $Me = 95$ ;  $SD = 17.029$ ).

To test whether the groups differed in the vividness of visual imagery, as measured by the VVIQ-2PL questionnaire, an independent samples t-test was performed. Significant differences between the groups were observed,  $t(18) = 16.364$ ;  $p < 0.001$ ;  $d = 7.318$  (Fig. 7B). Participants in the CG had a higher score ( $M = 122.60$ ;

SD=13.459) than AG ( $M=40.90$ ;  $SD=8.252$ ). The maximum score that can be obtained in the VVIQ-2PL is 160 points, while the minimum score is 32 points. In this study, a threshold of up to 64 points was used for AG. This is because in the literature to date, researchers have used the first version of the Visual Imagery Vividness Questionnaire VVIQ<sup>5</sup>, which has half as many statements, making the maximum possible score 80. We decided to double the minimum value of 16 from the VVIQ questionnaire and adapt this value to the VVIQ-2PL questionnaire as the qualifying threshold for the experimental group, in line with the findings of other researchers<sup>53,54</sup>.

It was also tested whether the two groups differed in kinesthetic imagery, external visual motor imagery and internal motor imagery, as measured by the MIQ-3 questionnaire. A t-test for independent samples was performed and significant differences were noted for internal motor imagery (IVI) (Fig. 7A),  $t(16)=4.368$ ;  $p<0.001$ ;  $d=2.072$ . Those in the CG scored higher ( $M=5.850$ ;  $SD=0.973$ ) than AG ( $M=3.313$ ;  $SD=1.487$ ). For external visual-motor imagery (EVI) (Fig. 7A), Levene's test showed significant differences,  $F(16)=6.054$ ;  $p=0.026$ , indicating a violation of the assumption of equality of variance. Therefore, a t-test for heterogeneous variances was performed, which showed significant differences between the groups,  $t(16)=3.561$ ;  $p=0.003$ ;  $d=1.689$ . Individuals in the CG scored higher ( $M=5.800$ ;  $SD=0.715$ ) than AG ( $M=3.375$ ;  $SD=2.013$ ). Finally, it was tested whether there were significant differences between the groups in KI (Fig. 7A). An independent sample t-test showed no significant differences between the groups,  $t(16)=0.762$ ;  $p=0.457$ .

### Stimulus for pupillary response task

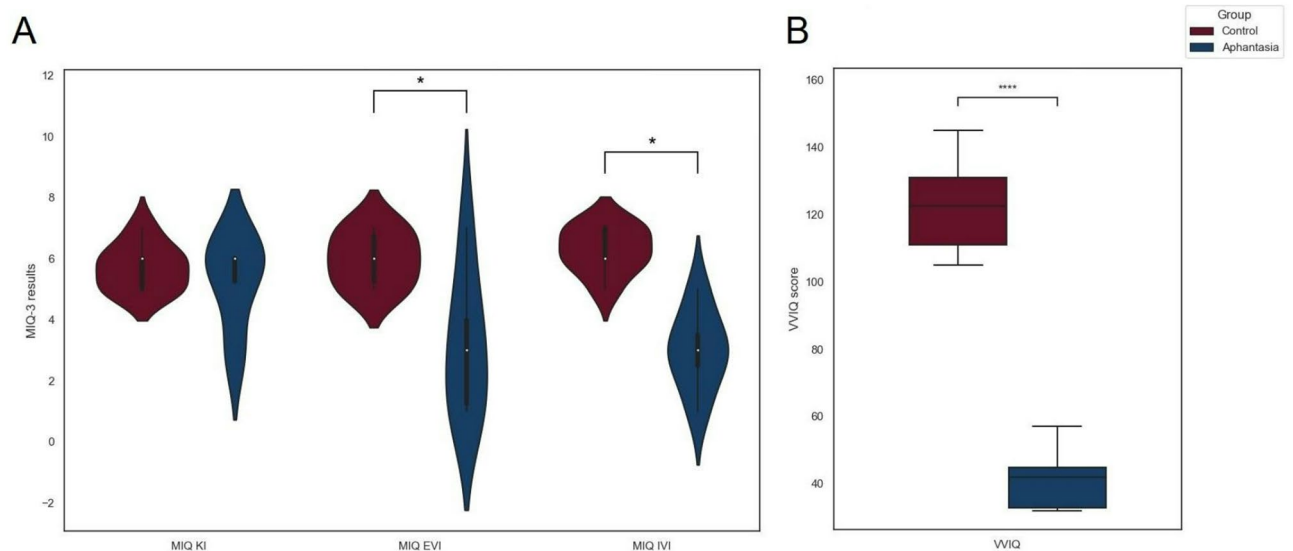
All stimuli were designed according to the article<sup>11</sup>. Thirty-two unique achromatic shape stimuli were constructed, representing 32 trials for each participant. They were equally divided based on a  $2 \times 2$  factorial model, under two luminance conditions ('light' or 'dark') and two set-size conditions ('set-size-one' or 'set-size-four'). The set-size-one consisted of an equilateral triangle whose diameter on the monitor was 131 mm at 12.5 degrees of angle. These were used to construct set-size-four stimuli, which consisted of four smaller equilateral triangles. The set-size-four stimuli had a visual angle of 10.8 degrees or 18.9 degrees, depending on the orientation. Each stimulus was oriented at one of the angles 0, 90, 180, 270 to ensure that none of the stimuli were repeated for the participant. The background on which the stimuli were presented was gray at 26 cd/m<sup>2</sup>.

### Apparature fNIRS

Relative changes in oxygenated (HbO) and deoxygenated (HbR) hemoglobin concentrations were measured using a functional near-infrared spectroscopy (Photon Cap, Cortivision sp. z.o.o, Poland) with continuous-wave method. The sampling frequency was 5 Hz. The 26 channels were located within the International EEG 10–5 system in the following areas: primary left motor cortex (LMC), primary right motor cortex (RMC), left middle frontal gyrus (L-MFG), right middle frontal gyrus (R-MFG) and primary visual cortex (VC) (Fig. 8). The primary visual cortex (VC) area was discarded due to the difficulty in obtaining a good signal quality. As a result, the signal was analyzed from 22 channels.

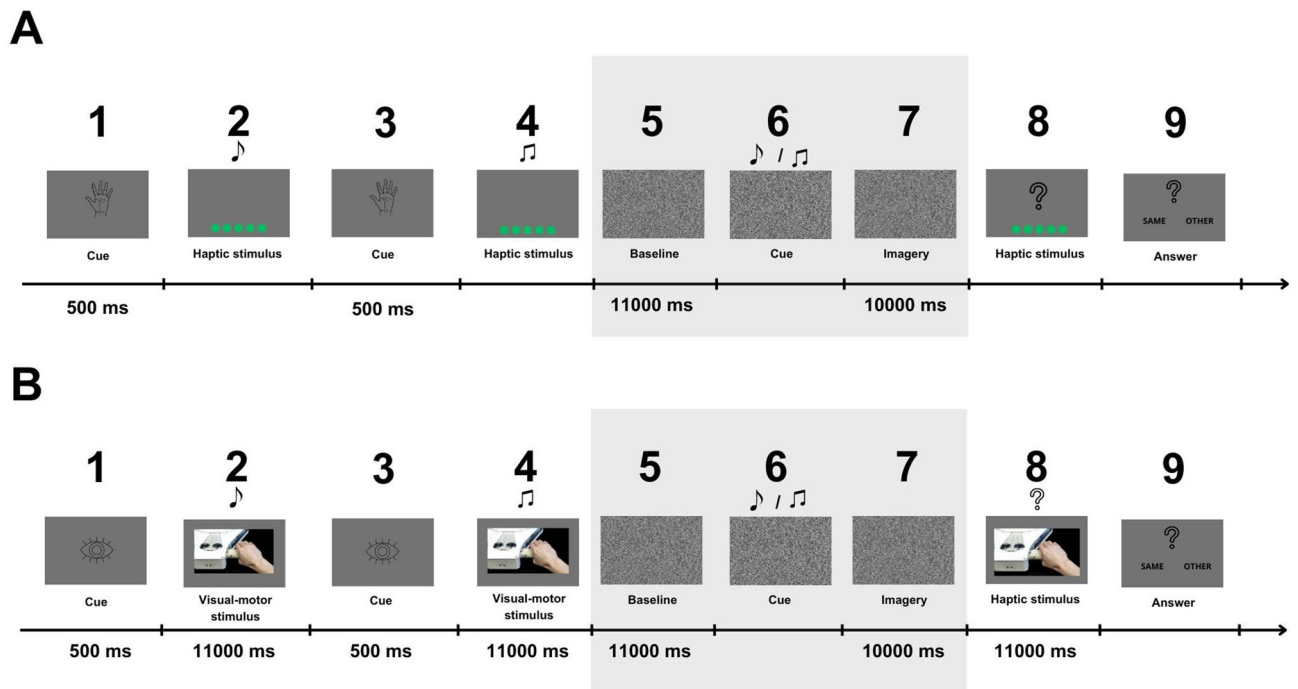
### Eye-tracking

The SMI RED500 sub-monitor eye-tracker (SensoMotoric Instruments GmbH) was used to measure pupil response. The sampling frequency was set at 500 Hz. The experimental procedure was presented on a 22" monitor with a screen resolution of 1680 × 1050 pixels. It was designed using E-Prime 3.0 software, and the



**Fig. 7.** Questionnaire results. **A.** MIQ-3 scores for three subscales (KI, EVI, IVI) between the AG and the CG. **B.** VVIQ scores between the AG and the CG.





**Fig. 9.** Experimental procedure. (A) Kinesthetic motor imagery task. (B) Visual-motor imagery task.

### Electromyography

During the experimental procedure, electromyographic (EMG) measurements were taken to control for unintentional hand movements. The signal was recorded using a Flex Comp Infinity device with two T3402 electrodes attached to the finger flexors.

## Experimental procedure

### Motor imagery task

The experimental procedure was designed in PsychoPy2 (version 1.82). Visual stimulus was presented at the 22" LCD monitor (Dell, Inc., Round Rock, TX, USA) with a screen resolution 1680 × 1050 pixels and refresh rate 60 Hz. Monitor was settled up in a 80 cm distance from a participant. Experimental procedure was adapted from Zapala et al.<sup>25</sup>. All participants performed both the kinesthetic motor imagery (KMI) and visual motor imagery (VMI) task (Fig. 9). The KMI condition consisted of squeezing the haptic interface handles, while the VMI consisted of observing a video in which the haptic interface handle was squeezed (detailed explanation below). Each participant completed 32 trials (16 for the KMI/VMI conditions). These trials were presented in random order. At the beginning of the study, the participant underwent a training procedure under the guidance of an experimenter who explained the whole experiment. Before the start of the recording, the participant was informed about the restriction of hand and body movements while imagining the seventh part of the experiment. It was also reported that the electrodes attached to the forearms were designed to measure muscle tension to ensure the participants followed the prescribed instructions.

The KMI task was divided into nine stages (see Fig. 9). In the first part, a hand symbol (500 ms) was displayed, informing the participant that they should prepare to tighten the grip of the device. In the second stage, the participant heard one of the sounds and then tightened the grip five times with their right hand. The grip offered different levels of resistance. By this, the participant learned the feeling of resistance corresponding to the sound. The command in the third stage corresponded to the first stage. In the fourth stage, the participant heard another sound and squeezed the handle five times, learning the feeling of resistance corresponding to the second sound. At the fifth stage, a white noise appeared, labelled in the procedure as a baseline. At this point, the participant had to be in a state of relaxation. In stage six, one of the previously presented sounds was emitted. This sound corresponds to the assigned resistance. In the seventh stage, the participant imagined the resistance that accompanied the sound they had just heard. In the eighth step, the participant tightened the control resistance five times. The sensation of movement could be the same as the one the participant had just imagined or different. In the ninth stage, a question ('Was the feeling during stage eight the same or different from what you imagined during stage seven?') and two answers ('same' or 'different') appeared on the screen. The participant was asked to select the correct answer by pressing the left or right button under the handles.

The VMI task was divided into nine parts. In the first part, an eye symbol was displayed (500 ms). This informed the participant that the viewing task was about to begin. In the second stage, the participant listened to one of the presented sounds and then watched a short video showing the movement of tightening the grip of the haptic interface. The participant was asked to associate the presented sound with the resistance associated with the observed grip. This activity was performed at different speeds, which the participant was asked to recognise.

The third phase was the same as the first. In the fourth phase, the participant heard another sound and then watched a short video, again showing the movement of squeezing the haptic interface. The participant did the same, attributing the presented sound to the resistance associated with the observed clenching. In the fifth stage, a white noise was presented, signaling the participant to relax. In the sixth step, one of the previously presented sounds corresponding to the assigned resistance was emitted. In the seventh step, the participant imagined the resistance associated with the sound just heard. In the eighth step, the participant watched the video again. The observed movement could have been the same as or different from the previous movement. The ninth stage was the same as the KMI task, except that this time the question was about the observed speed of the movement rather than the kinesthetic sensation. The participant responded by pressing the left or right button under the handles of the haptic interface.

### Pupillary response task

Procedure was adapted from Kay et al.<sup>11</sup>. Every trial has began with the presentation of a white fixation cross at the center of a gray screen for 1 s. Next, either one or four triangles of varying brightness was presented at the center of grey screen for 5 s. Participants were instructed to focus on the stimuli during this time and remember it's size, orientation and level of brightness. Consequently, a black screen with a cross of fixation was presented for 8 s. It allows, after-image to be faded and pupils to dilate back to equivalent resting levels. Then the gray screen was presented again for 6 s. Participant were instructed by single beep to start imaging the previously observed image during that trial. At the end, participants report the vividness of their imagery, by using 1–4 scale (1 being 'not vivid at all - no shape appeared in imagery'; 4 being 'very vivid - almost like seeing it') via keyboard.

### FNIRS signal processing

Data pre-processing was carried out using CortiPrism v1.3 software (Cortivision sp. z o. o., Poland, Lublin). Signal was analysed from 22 channels from 3 regions of interest: middle frontal gyrus (MFG), left motor cortex (LMC) and right motor cortex (RMC). First, the.snirf files were loaded with the raw data to the software. Then, changes in the intensity of light for two wavelengths (760 nm and 850 nm) for each channel were converted for optical density. Consequently, based on Scalp Coupling Index (SCI)<sup>55</sup> channels were rejected using threshold 0.7. After using SCI the number of channels allocated to each area is as follows: MFG (15 channels), LMC (12 channels), RMC (15 channels). In the next step, movement artifacts were corrected using temporal derivative distribution repair (TDDR) function<sup>56</sup>. Then, lowpass with a value of 0.2, was applied to remove the physiological signal from respiration and the heartbeat. Then short channel correction based on changing the influence of superficial layers of brain tissue was applied<sup>57</sup>. Data were converted for relative changes of oxygenated and deoxygenated hemoglobin using modified Beer-Lambert law (MBLL) taking into account a partial pathway factor with a constant value of 6 for each participant<sup>58</sup>. At the end correlation based signal improvement (CBSI) was used considering negative correlation between oxygenated and deoxygenated hemoglobin<sup>59</sup>.

Subsequently, the data was exported for analysis using a simple averaging method with a window of 0 to 10 s for imagery conditions. Next, statistical analysis was conducted using software such as JASP 0.19.0.0 and IBM SPSS version 29 (IBM Corp. Released 2022). Visualizations were generated using scripts written in Python 3, with the pandas<sup>60</sup>, matplotlib<sup>61</sup>, seaborn<sup>62</sup>, statannotations<sup>63</sup>, and os libraries.

### Eye-tracking preprocessing

The data was exported using SMI eye-tracking software. Trials that contained less than 50% of the observations were removed. Data were averaged for the experimental condition (Imagery or Perception). The pupil averages during imaging were then corrected for baseline (pupil averages during imagery or perception were subtracted from baseline<sup>64</sup>).

### EMG preprocessing

The data were processed using a script written in MATLAB 2021b. The raw data were imported into EEGLAB 2022.0 software, after which conversion to root mean square was conducted. Thereafter, the signal was segmented from 0 to 10 s. Subsequently, the average for each window of the experimental condition from 0 to 10 s was counted. The signal was then analyzed in JASP 0.19.0.0 statistical software.

### Data availability

The datasets generated during the current study are available in the OSF repository. <https://osf.io/dk3m6/>.

Received: 16 December 2024; Accepted: 12 September 2025

Published online: 17 October 2025

### References

- Keogh, R., Pearson, J. & Zeman, A. Chapter 15 - Aphantasia: The science of visual imagery extremes, Handbook of Clinical Neurology, Elsevier. (2021). <https://doi.org/10.1016/B978-0-12-821377-3.00012-X>
- Knowles, L., Jones, K. & Zeman, A. Acquired aphantasia in 88 cases: A preliminary report. *J. Neurol. (Neurosurgery Psychiatry)*. **92** (8), A6–A7. <https://doi.org/10.1136/jnnp-2021-BNPA.17> (2021).
- Dawes, A., Keogh, R., Andriillon, T. & Pearson, J. A cognitive profile of multi-sensory imagery, memory and dreaming in aphantasia. *Sci. Rep.* **10**, 10022. <https://doi.org/10.1038/s41598-020-65705-7> (2020).
- Dawes, A. J., Keogh, R. & Pearson, J. Multisensory subtypes of aphantasia: mental imagery as supramodal perception in reverse. *Neurosci. Res.* **201**, 50–59. <https://doi.org/10.1016/j.neures.2023.11.009> (2024).
- Marks, F. D Vividness of visual imagery questionnaire (VVIQ). *APA PsycTests*. <https://doi.org/10.1037/t05959-000> (1973).
- Spector, P. E. Using Self-Report questionnaires in OB research: A comment on the use of a controversial method. *J. Organizational Behav.* **15** (5), 385–392 (1994).

7. Blomkvist, A. & Marks, D. F. Defining and ‘diagnosing’ aphantasia: condition or individual difference? *Cortex* **169**, 220–234. <https://doi.org/10.1016/j.cortex.2023.09.004> (2023).
8. Keogh, R. & Pearson, J. The blind mind: no sensory visual imagery in aphantasia. *Cortex* **105**, 53–60 (2018).
9. Keogh, R. & Pearson, J. Attention driven Phantom vision: measuring the sensory strength of attentional templates and their relation to visual mental imagery and aphantasia. *Philosophical Trans. Royal Soc. B: Biol. Sci.* **376** (1817), 20190688. <https://doi.org/10.1098/rstb.2019.0688> (2020).
10. Keogh, R. & Pearson, J. Revisiting the blind mind: still no evidence for sensory visual imagery in individuals with aphantasia. *Neurosci. Res.* **201**, 27–30. <https://doi.org/10.1016/j.neures.2024.01.008> (2024).
11. Kay, L., Keogh, R., Andrillon, T. & Pearson, J. The pupillary light response as a physiological index of aphantasia, sensory and phenomenological imagery strength. *eLife* **11** <https://doi.org/10.7554/eLife.72484> (2022).
12. Joshi, S. & Gold, J. I. Pupil size as a window on neural substrates of cognition. *Trends Cogn. Sci.* **24** (6), 466–480. <https://doi.org/10.1016/j.tics.2020.03.005> (2020).
13. Mathôt, S. Pupillometry: psychology, physiology, and function. *J. Cognition.* **1** (1), 16. <https://doi.org/10.5334/joc.18> (2018).
14. Barbur, J. L., Harlow, A. J. & Sahraie, A. Pupillary responses to stimulus structure, colour and movement. *Ophthalmic Physiol. Opt.* **12** (2), 137–141. <https://doi.org/10.1111/j.1475-1313.1992.tb00276.x> (1992).
15. Onorati, F., Barbieri, R., Mauri, M., Russo, V. & Mainardi, L. Characterization of affective States by pupillary dynamics and autonomic correlates. *Front. Neuroeng.* <https://doi.org/10.3389/fneng.2013.00009> (2013). 6.
16. Einhäuser, W. The Pupil as Marker of Cognitive Processes. In Q. Zhao (Ed.), *Computational and Cognitive Neuroscience of Vision* (pp. 141–169). Springer. (2017). [https://doi.org/10.1007/978-981-10-0213-7\\_7](https://doi.org/10.1007/978-981-10-0213-7_7)
17. Knight, K. F., Milton, F. & Zeman, A. Z. J. Memory without Imagery: No Evidence of Visual Working Memory Impairment in People with Aphantasia. *Proceedings of the Annual Meeting of the Cognitive Science Society*, 44(44). (2022). <https://escholarship.org/uc/item/0b16s06v>
18. Pounder, Z. et al. Individuals with congenital aphantasia show no significant neuropsychological deficits on imagery-related memory tasks. (2021). <https://doi.org/10.31234/osf.io/gqayt>
19. Milton, F. et al. Behavioral and neural signatures of visual imagery vividness extremes: aphantasia versus hyperphantasia. *Cereb. Cortex Commun.* **2** (2). <https://doi.org/10.1093/texcom/tgab035> (2021).
20. Decety, J. The neurophysiological basis of motor imagery. *Behav. Brain. Res.* **77** (1), 45–52. [https://doi.org/10.1016/0166-4328\(95\)00225-1](https://doi.org/10.1016/0166-4328(95)00225-1) (1996).
21. Stinear, C. M., Byblow, W. D., Steyvers, M., Levin, O. & Swinnen, S. P. Kinesthetic, but not visual, motor imagery modulates corticomotor excitability. *Exp. Brain Res.* **168** (1), 157–164. <https://doi.org/10.1007/s00221-005-0078-y> (2006).
22. Dupont, W., Papaxanthis, C., Madden-Lombardi, C. & Lebon, F. Explicit and implicit motor simulations are impaired in individuals with aphantasia. *BioRxiv* <https://doi.org/10.1101/2022.12.15.520602> (2022).
23. Lorey, B. et al. The embodied nature of motor imagery: the influence of posture and perspective. *Exp. Brain Res.* **194** (2), 233–243. <https://doi.org/10.1007/s00221-008-1693-1> (2009).
24. Vokeley, K. & Fink, G. R. Neural correlates of the first-person-perspective. *Trends Cogn. Sci.* **7** (1), 38–42. [https://doi.org/10.1016/S1364-6613\(02\)00003-7](https://doi.org/10.1016/S1364-6613(02)00003-7) (2003).
25. Zapala, D., Augustynowicz, P., Tokovarov, M., Iwanowicz, P. & Drozdziel, P. Brief visual deprivation effects on brain oscillations during kinesthetic and visual-motor imagery. *Neuroscience* **532**, 37–49. <https://doi.org/10.1016/j.neuroscience.2023.08.022> (2023).
26. Li, R. et al. Concurrent fNIRS and EEG for brain function investigation: A systematic, Methodology-Focused review. *Sensors* **22** (15). <https://doi.org/10.3390/s22155865> (2022). Article 15.
27. Tak, S. & Ye, J. C. Statistical analysis of fNIRS data: A comprehensive review. *NeuroImage* **85**, 72–91. <https://doi.org/10.1016/j.neuroimage.2013.06.016> (2014).
28. Strangman, G., Culver, J. P., Thompson, J. H. & Boas, D. A. A quantitative comparison of simultaneous BOLD fMRI and NIRS recordings during functional brain activation. *NeuroImage* **17** (2), 719–731. <https://doi.org/10.1006/nimg.2002.1227> (2002).
29. Jeannerod, M. The representing brain: neural correlates of motor intention and imagery. *Behav. Brain Sci.* **17** (2), 187–202 (1994).
30. Lorenc, E. S., Sreenivasan, K. K., Nee, D. E., Vandenbroucke, A. R. E. & D’Esposito, M. Flexible coding of visual working memory representations during distraction. *J. Neurosci.* **38** (23), 5267–5276. <https://doi.org/10.1523/JNEUROSCI.3061-17.2018> (2018).
31. Japee, S., Holiday, K., Satyshur, M. D., Mukai, I. & Ungerleider, L. G. A role of right middle frontal gyrus in reorienting of attention: A case study. *Front. Syst. Neurosci.* **9**. <https://doi.org/10.3389/fnsys.2015.00023> (2015).
32. Buckner, R. L., Raichle, M. E. & Petersen, S. E. Dissociation of human prefrontal cortical areas across different speech production tasks and gender groups. *J. Neurophysiol.* **74** (5), 2163–2173. <https://doi.org/10.1152/jn.1995.74.5.2163> (1995).
33. Cappa, S. F., Sandrini, M., Rossini, P. M., Sosta, K. & Miniussi, C. The role of the left frontal lobe in action naming. *Neurology* **59** (5), 720–723. <https://doi.org/10.1212/WNL.59.5.720> (2002).
34. Agcaoglu, O., Miller, R., Mayer, A. R., Hugdahl, K. & Calhoun, V. D. Lateralization of resting state networks and relationship to age and gender. *NeuroImage* **104**, 310–325. <https://doi.org/10.1016/j.neuroimage.2014.09.001> (2015).
35. Sabaté, M., González, B. & Rodríguez, M. Brain lateralization of motor imagery: motor planning asymmetry as a cause of movement lateralization. *Neuropsychologia* **42** (8), 1041–1049. <https://doi.org/10.1016/j.neuropsychologia.2003.12.015> (2004).
36. Meng, M., Chang, S., Zhang, X. & Pearson, J. Imageless imagery in aphantasia: decoding non-sensory imagery in aphantasia. (2023). <https://doi.org/10.21203/rs.3.rs-3162223/v1>
37. Corbetta, M., Patel, G. & Shulman, G. L. The reorienting system of the human brain: from environment to theory of Mind. *Neuron* **58** (3), 306–324. <https://doi.org/10.1016/j.neuron.2008.04.017> (2008).
38. Friedman-Hill, S. R., Robertson, L. C., Desimone, R. & Ungerleider, L. G. Posterior parietal cortex and the filtering of distractors. *Proc. Natl. Acad. Sci.* **100** (7), 4263–4268. <https://doi.org/10.1073/pnas.0730772100> (2003).
39. Weissman, D. H. & Prado, J. Heightened activity in a key region of the ventral attention network is linked to reduced activity in a key region of the dorsal attention network during unexpected shifts of Covert visual Spatial attention. *NeuroImage* **61** (4), 798–804. <https://doi.org/10.1016/j.neuroimage.2012.03.032> (2012).
40. Pearson, J. The human imagination: the cognitive neuroscience of visual mental imagery. *Nat. Rev. Neurosci.* **20** (10), 624–634 (2019).
41. Guillot, A. et al. Brain activity during visual versus kinesthetic imagery: an fMRI study. *Hum. Brain. Mapp.* **30** (7), 2157–2172 (2009).
42. Neuper, C., Scherer, R., Reiner, M. & Pfurtscheller, G. Imagery of motor actions: differential effects of kinesthetic and visual-motor mode of imagery in single-trial EEG. *Cogn. Brain. Res.* **25** (3), 668–677 (2005).
43. Kilintari, M., Narayana, S., Babajani-Feremi, A., Rezaie, R. & Papanicolaou, A. C. Brain activation profiles during kinesthetic and visual imagery: an fMRI study. *Brain Res.* **1646**, 249–261 (2016).
44. Abrahams, S. et al. Functional magnetic resonance imaging of verbal fluency and confrontation naming using compressed image acquisition to permit overt responses. *Hum. Brain. Mapp.* **20** (1), 29–40. <https://doi.org/10.1002/hbm.10126> (2003).
45. Brown, S., Martinez, M. J. & Parsons, L. M. Music and Language side by side in the brain: A PET study of the generation of melodies and sentences. *Eur. J. Neurosci.* **23** (10), 2791–2803. <https://doi.org/10.1111/j.1460-9568.2006.04785.x> (2006).
46. Reeder, R. R., Pounder, Z., Figueroa, A., Jüllig, A. & Azañón, E. Non-visual Spatial strategies are effective for maintaining precise information in visual working memory. *Cognition* **251**, 105907. <https://doi.org/10.1016/j.cognition.2024.105907> (2024).
47. Sheehan, P. W. A shortened form of betts’ questionnaire upon mental imagery. *J. Clin. Psychol.* **23** (3), 386–389. [https://doi.org/10.1002/1097-4679\(196707\)23:3](https://doi.org/10.1002/1097-4679(196707)23:3) (1967).

48. Kreidler, S. M., Muller, K. E., Grunwald, G., Ringham, B. M., Coker-Dukowitz, Z. T., Sakhadeo, U. R., ... Glueck, D. H. (2013). GLIMMMPSE: online power computation for linear models with and without a baseline covariate. *Journal of statistical software*, 54, 1–26.
49. Klein, F., Debener, S., Witt, K. & Kranczioch, C. fMRI-based validation of continuous-wave fNIRS of supplementary motor area activation during motor execution and motor imagery. *Sci. Rep.* **12** (1), 3570 (2022).
50. Oldfield, R. C. The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia* **9**, 97–113. [https://doi.org/10.1016/0028-3932\(71\)90067-4](https://doi.org/10.1016/0028-3932(71)90067-4) (1971).
51. Budnik-Przybylska, D., Szczypińska, M. & Karasiewicz, K. Reliability and validity of the Polish version of the movement imagery Questionnaire-3 (MIQ-3). *Curr. Issues Personality Psychol.* **4** (4), 253–267 (2016).
52. Jankowska, D. M. & Karwowski, M. *How Vivid Is your Mental Imagery?* (European Journal of Psychological Assessment, 2022).
53. Dance, C. J., Ipser, A. & Simner, J. The prevalence of aphantasia (imagery weakness) in the general population. *Conscious. Cogn.* **97**, 103243. <https://doi.org/10.1016/j.concog.2021.103243> (2022).
54. Takahashi, J. et al. Diversity of aphantasia revealed by multiple assessments of visual imagery, multisensory imagery, and cognitive style. *Front. Psychol.* **14**, 1174873. <https://doi.org/10.3389/fpsyg.2023.1174873> (2023).
55. Pollonini, L., Bortfeld, H. & Oghalai, J. S. PHOEBE: a method for real time mapping of optodes-scalp coupling in functional near-infrared spectroscopy. *Biomedical Opt. Express.* **7** (12), 5104–5119 (2016).
56. Fishburn, F. A., Ludlum, R. S., Vaidya, C. J. & Medvedev, A. V. Temporal derivative distribution repair (TDDR): a motion correction method for fNIRS. *Neuroimage* **184**, 171–179 (2019).
57. Gagnon, L. et al. Short separation channel location impacts the performance of short channel regression in NIRS. *NeuroImage* **59** (3), 2518–2528. <https://doi.org/10.1016/j.neuroimage.2011.08.095> (2012).
58. Whiteman, A. C., Santosa, H., Chen, D. E., Perlman, S. B. & Huppert, T. Investigation of the sensitivity of functional near-infrared spectroscopy brain imaging to anatomical variations in 5- to 11-year-old children. *Neurophotonics* **5** (1), 011009. <https://doi.org/10.1117/1.NPh.5.1.011009> (2017).
59. Cui, X., Bray, S. & Reiss, A. L. Functional near infrared spectroscopy (NIRS) signal improvement based on negative correlation between oxygenated and deoxygenated hemoglobin dynamics. *NeuroImage* **49** (4), 3039–3046. <https://doi.org/10.1016/j.neuroimage.2009.11.050> (2010).
60. McKinney, W. Pandas: a foundational python library for data analysis and statistics. *Python High. Perform. Sci. Comput.* **14** (9), 1–9 (2011).
61. Barrett, P., Hunter, J., Miller, J. T., Hsu, J. C. & Greenfield, P. matplotlib—A Portable Python Plotting Package. In *Astronomical data analysis software and systems XIV* (Vol. 347, p. 91). (2005), December.
62. Waskom, M. L. Seaborn: statistical data visualization. *J. Open. Source Softw.* **6** (60), 3021 (2021).
63. Charlier, F. Trevismd/statannotations [Python]. <https://github.com/trevismd/statannotations> (2024). (Original work published 2020).
64. Mathôt, S., Fabius, J., Van Heusden, E. & Van der Stigchel, S. Safe and sensible preprocessing and baseline correction of pupil-size data. *Behav. Res. Methods.* **50** (1), 94–106. <https://doi.org/10.3758/s13428-017-1007-2> (2018).

## Author contributions

R.K, D.Z, P.A designed the study R.K performed the experiment and collected the data. R.K, D.Z and P.A analyzed the fNIRS, EMG and pupil data. R.K, D.Z and P.A performed statistical analysis, R.K interpreted the results. R.K, D.Z, M.S drafted the manuscript M.S supervised the study.

## Declarations

## Competing interests

The authors declare no competing interests.

## Additional information

**Supplementary Information** The online version contains supplementary material available at <https://doi.org/10.1038/s41598-025-20168-6>.

**Correspondence** and requests for materials should be addressed to R.K.

**Reprints and permissions information** is available at [www.nature.com/reprints](http://www.nature.com/reprints).

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

**Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

© The Author(s) 2025