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Visual-Spatial updating and cortical excitability during vestibular stimulation

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Background

Nystagmus = rhythmic involuntary oscillation of the eyes



Oscillopsia = illusion of a perceived unstable visual world



Nystagmus

Acquired

- Damage to the peripheral vestibular organ or vestibulo-cochlear nerve
 - Trauma
 - Inflammation
 - Tumor
- Damage to the vestibular nuclei in the brainstem
 - multiple sclerosis
 - stroke
 - microangiopathy

Debilitating oscillopsia for days - months

Congenital

Infantile Nystagmus Syndrome

- Early onset forms observed after the age of 6 months
- No clear defect of the vestibular or visual system

Usually no complaint of oscillopsia

Theories for oscillopsia suppression:

- A) Efference copy through extra retinal eye movement signal** (*Abadi et al., 1999; Leigh et al., 1988; Dell'Osso, 2011; Goldstein et al., 1992*)
- B) Foveation period sampling during slow movements** (*Abadi and Worfolk, 1989; Van Der Steen et al., 1992*)
 - probably less relevant in relatively fast nystagmus movements
- C) Elevated threshold of visual motion cortex (V5)** (*Dieterich and Brandt, 1987; Shallo-Hoffmann et al., 1998*)
- D) Combination of A-C depending on underlying pathophysiology**

Aim and hypothesis

Investigate the neural mechanisms of oscillopsia suppression focusing on modulation of visual cortical excitability in visual area V5 and the efference copy dependent mechanisms of visual cortical spatial updating

- A) cortical excitability of V5 depends on the velocity of an evoked nystagmus**
- B) visual spatial updating will be absent during vestibular stimulation compared to no stimulation**

Setup

Healthy participants
(N=16; M:12,F:4)

Recording of eye position and
nystagmus velocity with EOG

Stimulation of visual phosphenes
using single pulse TMS over V5
(3 cm above, 5 cm lateral toinion)

Phosphene location collected
via touch screen input

Vestibular stimulation using
caloric irrigation (ICS MEDICAL)

Irrigation(45s)
400ml/min → TMS(7s Delay)
10s Interval → Repeat block
X5

TMS- evoked phosphenes

Illusory percept of light, caused by mechanical, electrical, or chemical stimulation of the retina or visual cortex

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(N=16; M:12,F:4)

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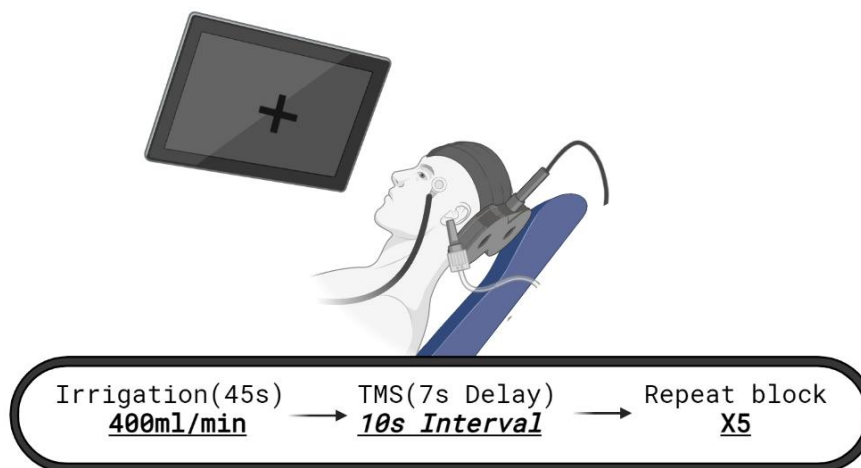
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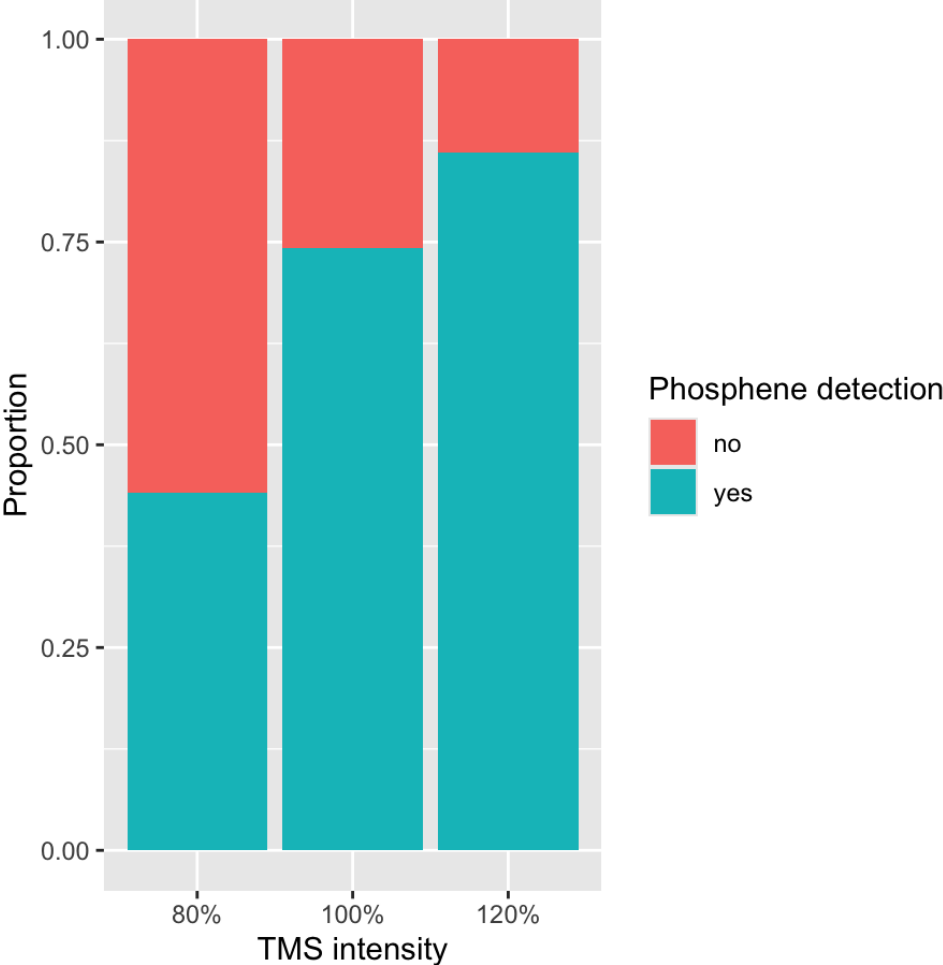
A) Cortical V5 excitability

- Initial personal threshold determined via 50% registered phosphenes
- 5 blocks of 18 TMS pulses (90 pulses / subject)
- Intensity of 80%, 100% and 120% of personal threshold, randomly distributed
- Touch or oral report if phosphene was seen on screen

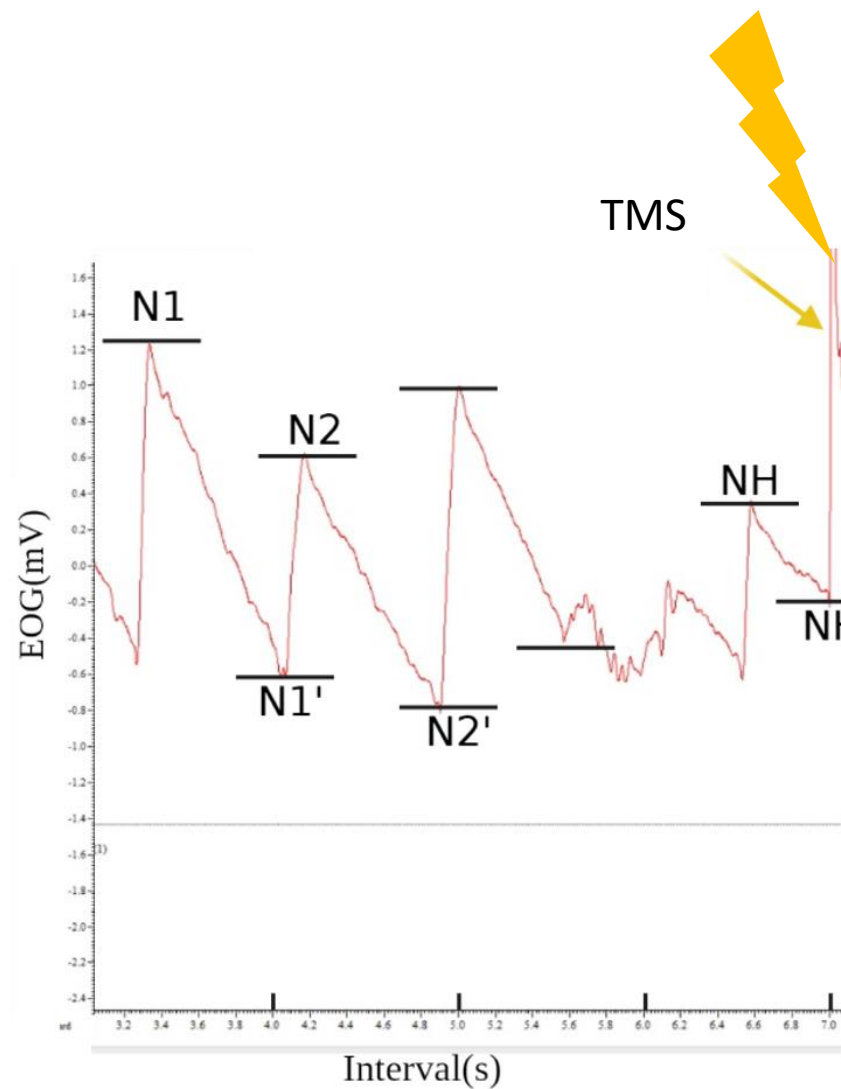
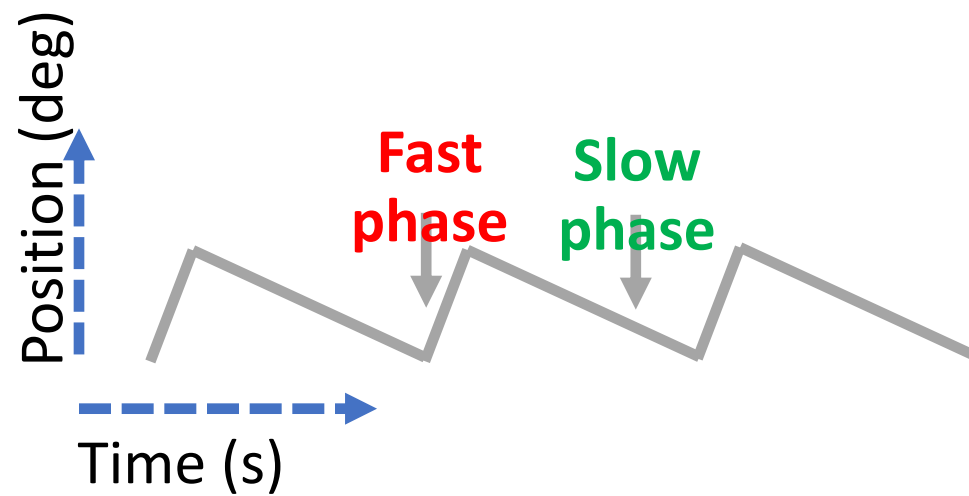


A) Cortical V5 excitability

- Overall phosphene detection



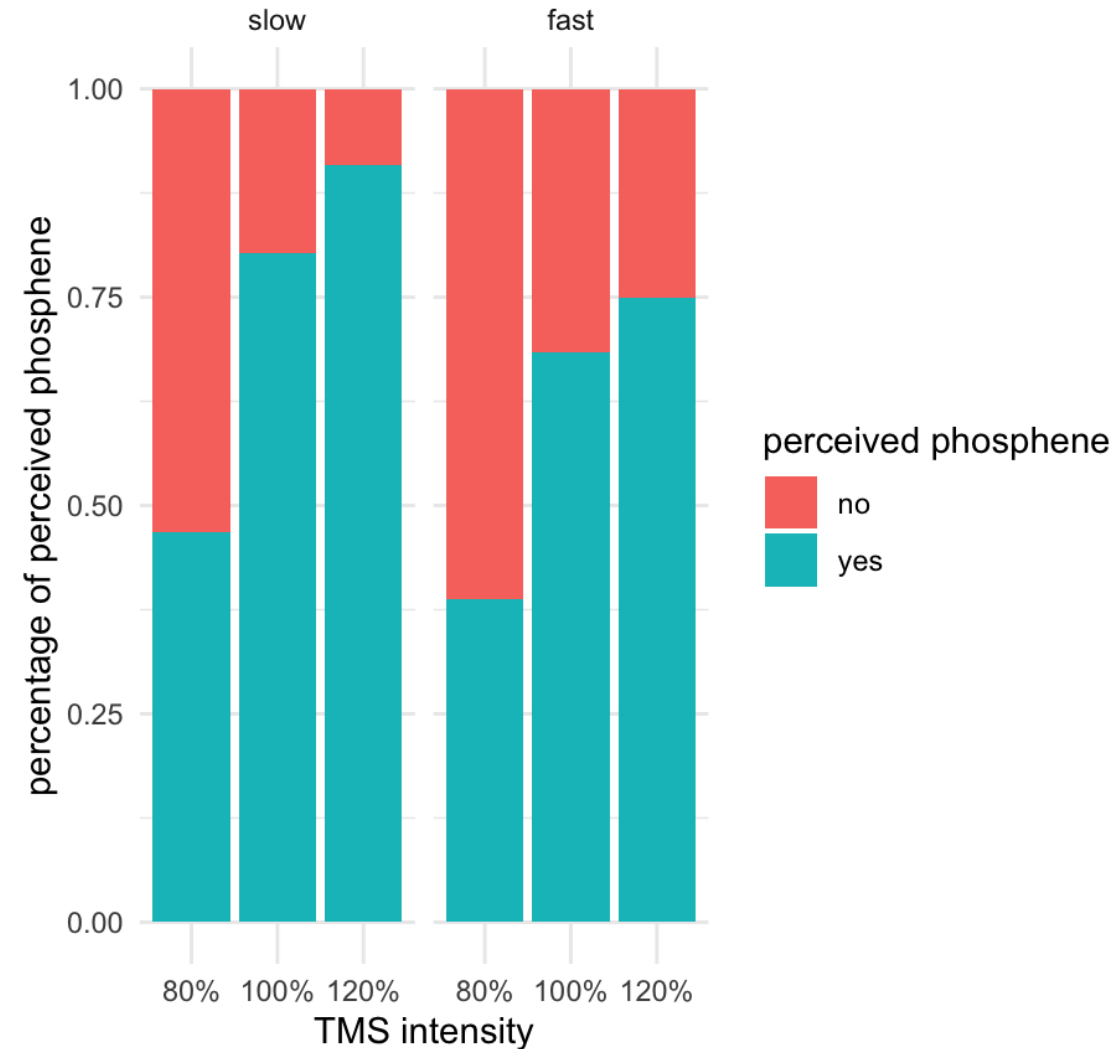
Eye position tracking



A) Cortical V5 excitability

- Slow phase vs phase vs nystagmus

<i>Predictors</i>	Phosphene detection		
	<i>Odds Ratios</i>	<i>CI</i>	<i>p</i>
nystagmus phase [fast vs. slow]	0.52	0.34 – 0.79	0.002
TMS intensity [80% vs 100%]	0.21	0.13 – 0.32	<0.001
TMS intensity [120% vs 100%]	2.03	1.20 – 3.43	0.009

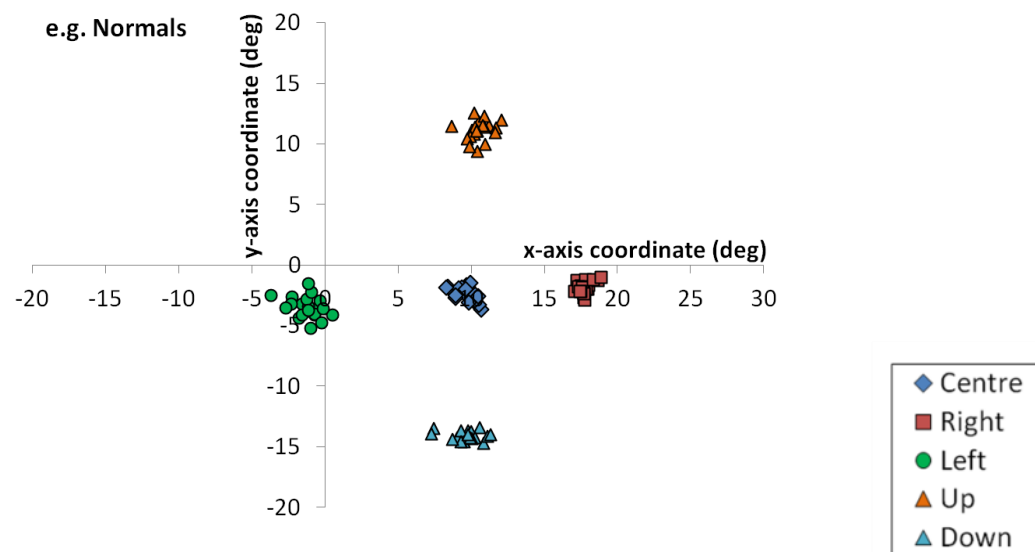


- A) cortical excitability of V5 depends on the velocity of an evoked nystagmus**
- B) visual spatial updating will be absent during vestibular stimulation**



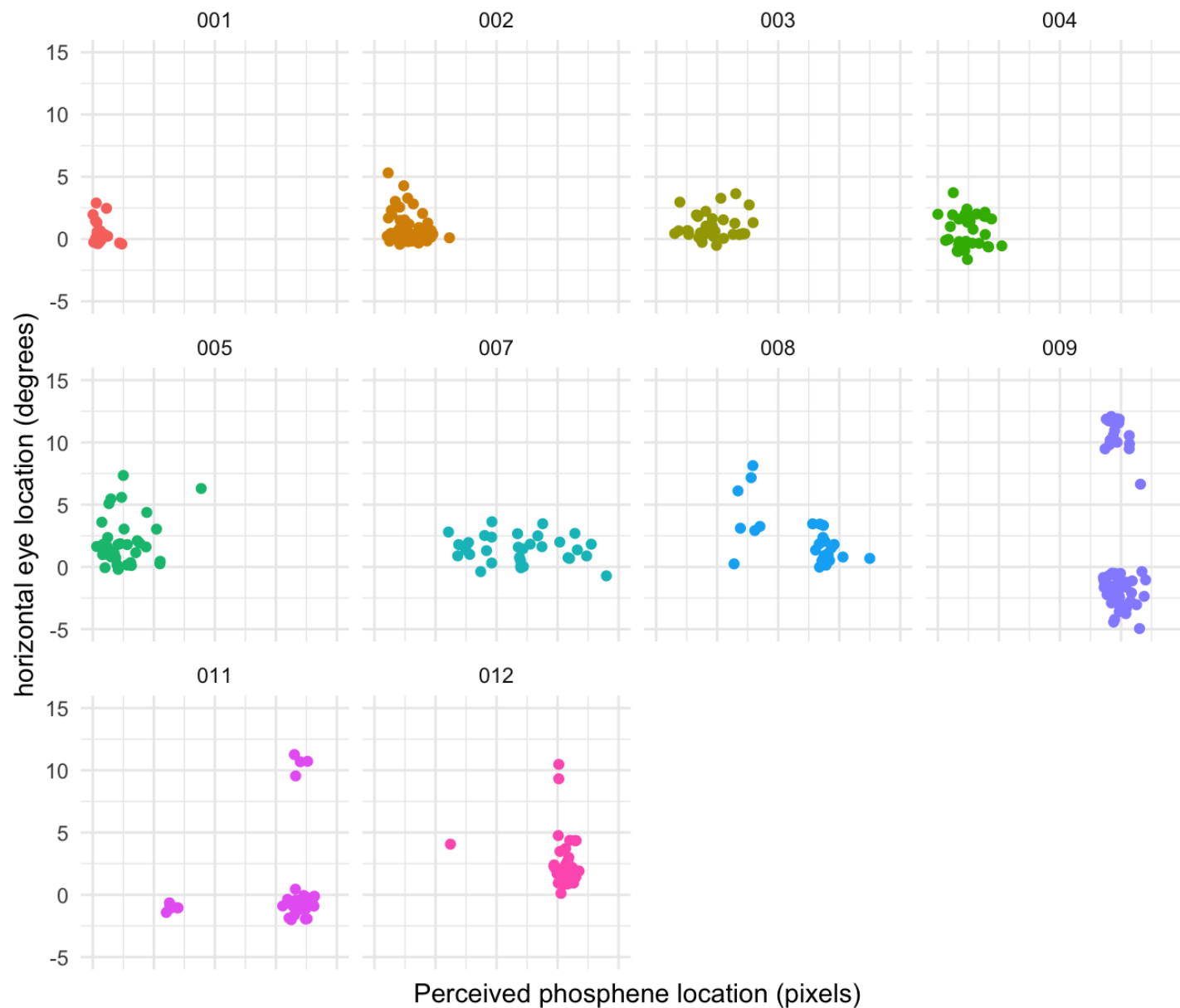
B) Visuo-spatial updating

- Retinotopic property of V5 phosphenes without vestibular stimulation



B) Visuo-spatial updating

- No correlation between perceived phosphene location and eye position during vestibular stimulation
- data suggests loss of visual-spatial updating during induced nystagmus

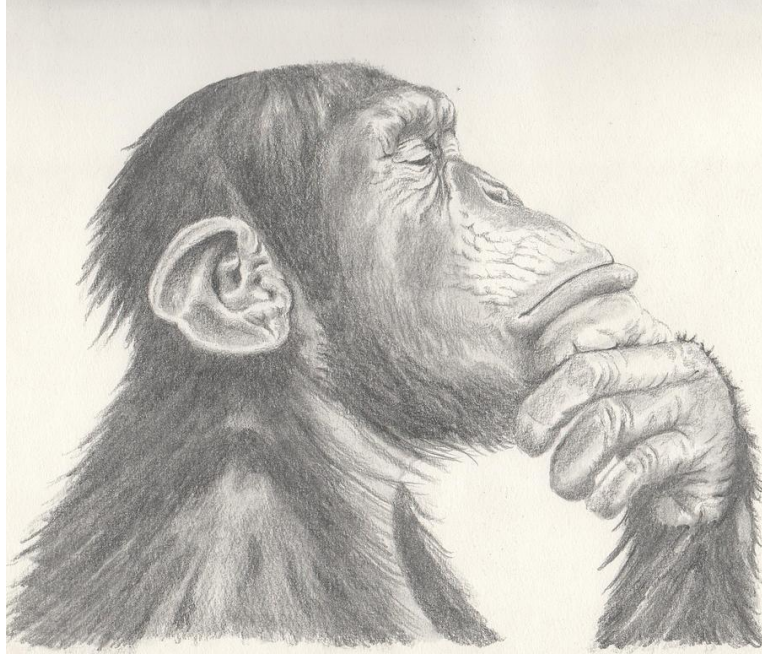


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Outlook

- Testing in INS subject, preferably those with no oscillopsia
- Possibly testing patients with oscillopsia (MS?)
- Improve eye location tracking



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Dominik Straumann



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