physiology, function and physics of the vestibular system“

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can we learn from what happens if something goes wrong in the vestibular system ?

acute but **transient** symptoms

acute <u>unilateral</u> loss or fluctuating function (neuritis, Ménière…)

- acute severe vertigo, severe nausea, falling and imbalance (the classical leading symptoms for diagnosis)

acute <u>bilateral</u> loss

- acute severe intolerance to head movements, nausea and imbalance (no vertigo: so the diagnosis is often missed)

poor dynamic compensation: sustained

- impact on various autonomic functions
- reduced automatisation of balance
- reduced dynamic visual acuity
- reduced perception of self motion
- hypersensitivity for optokinetic stimuli
- reduced ability to discriminate between self-motion and environmental motion
- secondary: fear and fatigue (cognitive load)

which complaints are related to vestibular deficits ?

which complaints are related to natural limitations ?

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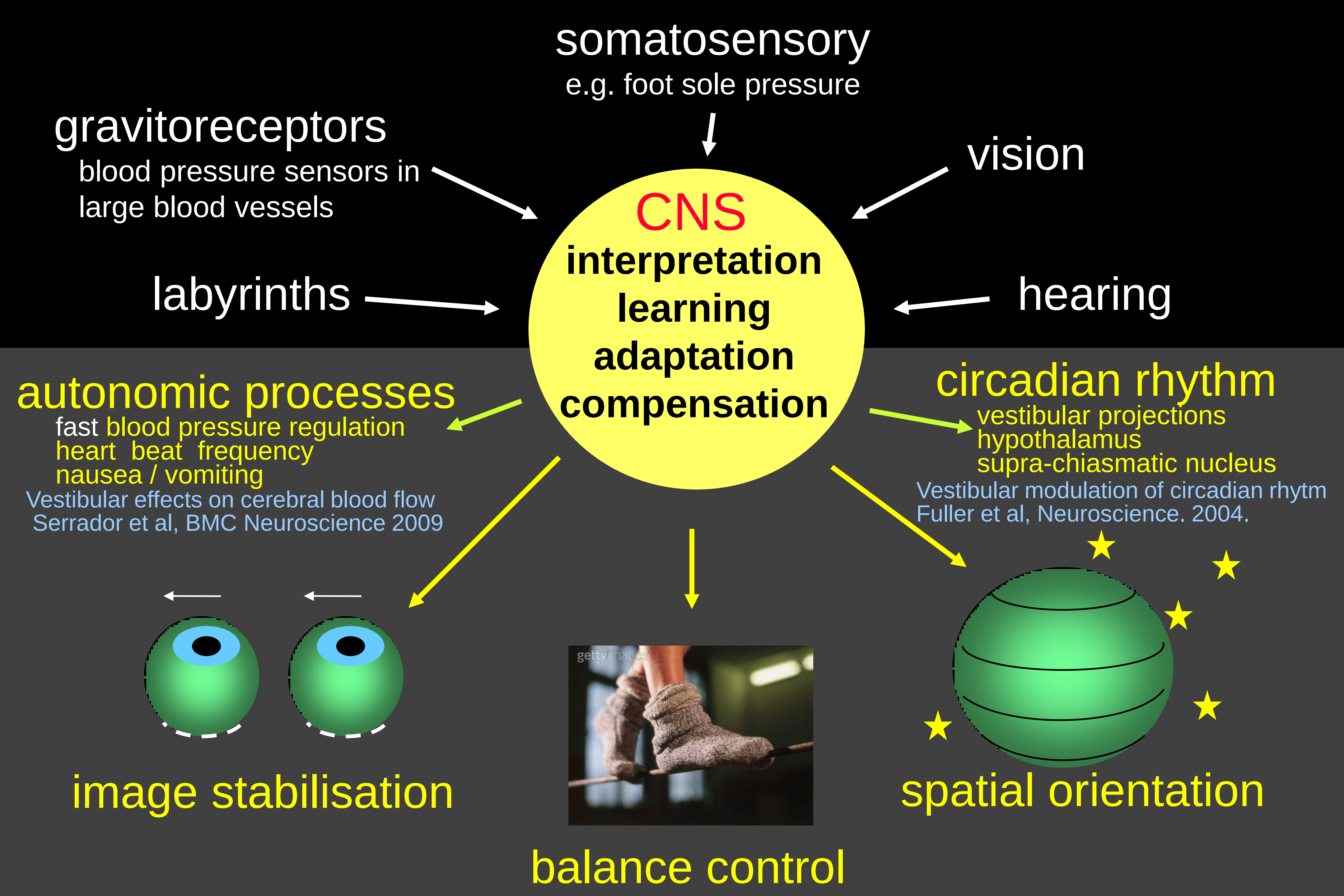

somatosensory
e.g. foot sole pressure
gravitoreceptors
blood pressure sensors in large blood vessels
vision
CNS
interpretation
learning
adaptation
compensation
labyrinths
hearing
autonomic processes
fast blood pressure regulation
heart beat frequency
nausea / vomiting
Vestibular effects on cerebral blood flow
Serrador et al, BMC Neuroscience 2009
circadian rhythm
vestibular projections
hypothalamus
supra-chiasmatic nucleus
Vestibular modulation of circadian rhytm
Fuller et al, Neuroscience. 2004.
gettyimages
image stabilisation
balance control
spatial orientation

complaints related to vestibular dysfunction

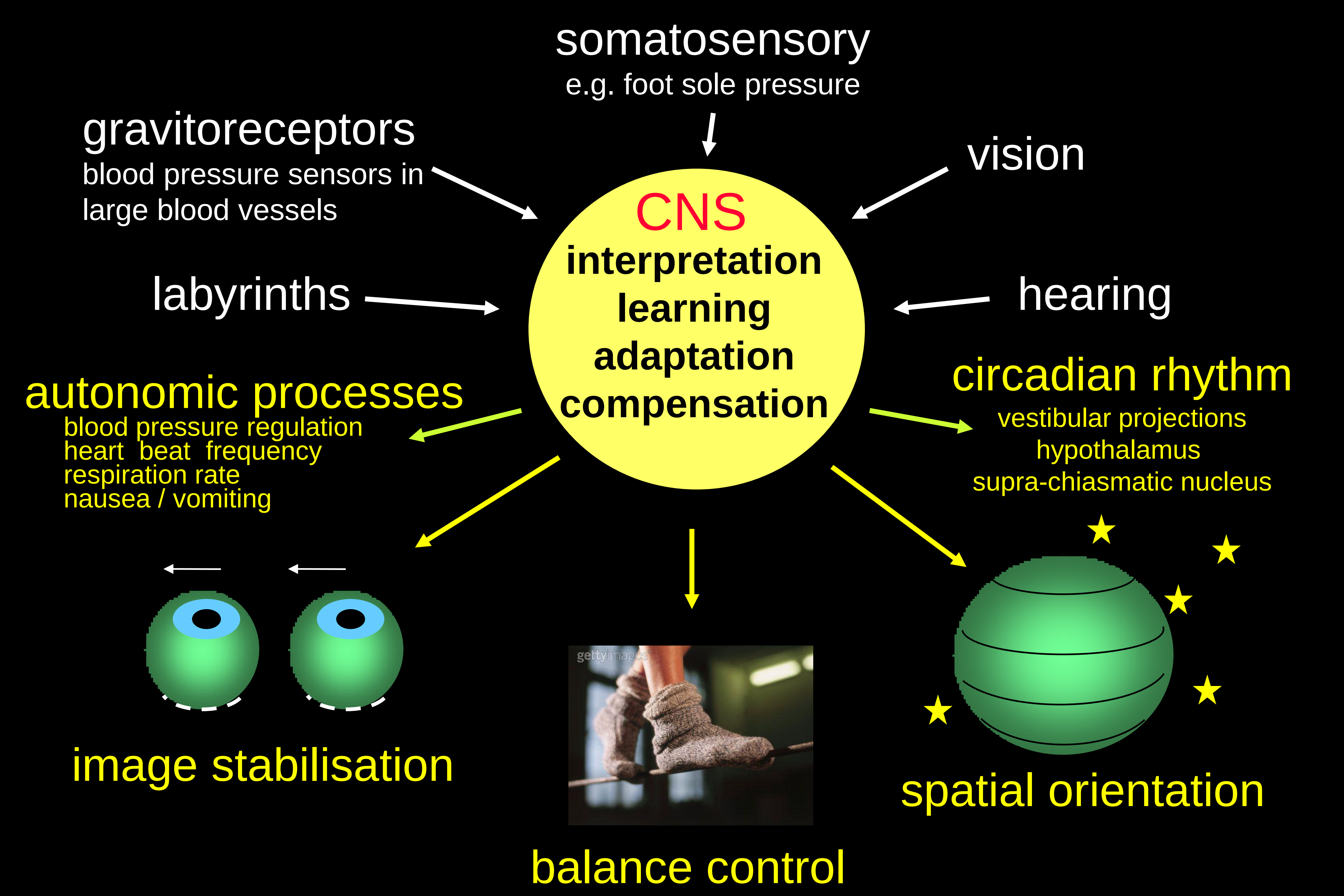
acute loss or fluctuating function

transient: vertigo, nausea, falling / imbalance

remaining peripheral vestibular function loss

sustained:

- enhanced neuro-vegetative sensitivity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balance control

complaints related to vestibular dysfunction

acute loss or fluctuating function

transient: vertigo, nausea, falling / imbalance

remaining peripheral vestibular function loss

sustained:

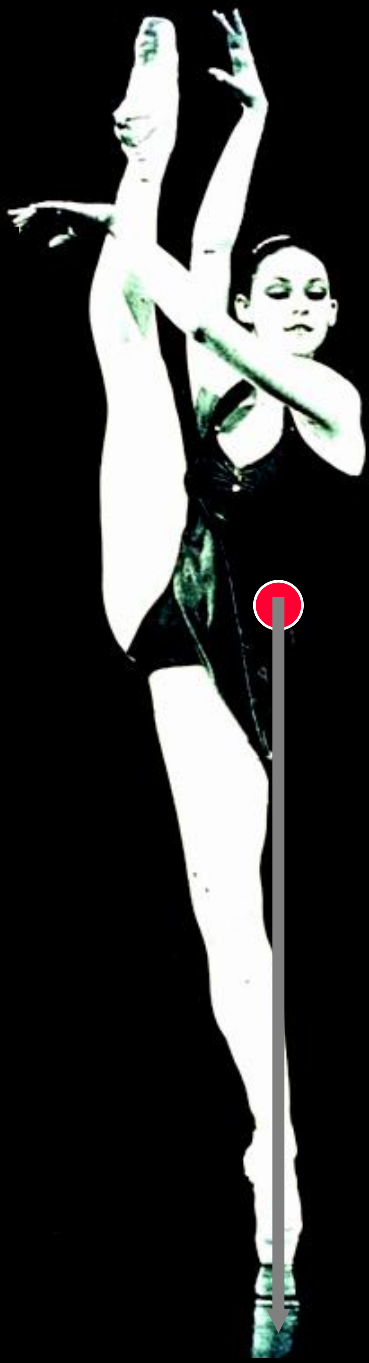
- reduced perception of self motion
- hypersensitivity for optokinetic stimuli
- reduced ability to discriminate between self-motion and environmental motion

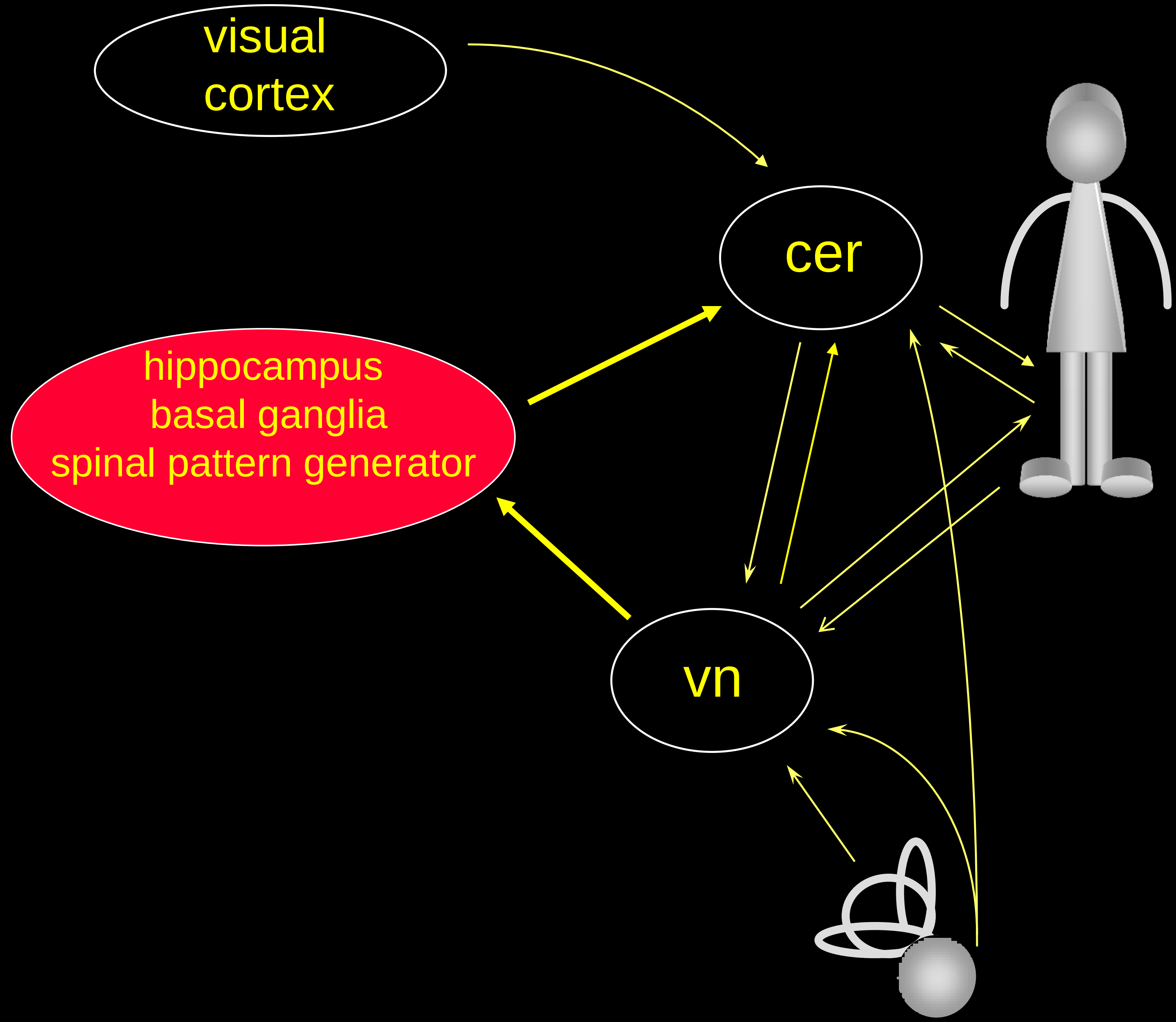
vestibular impact upon postural control

- regulation of muscle tone
  relative to gravity

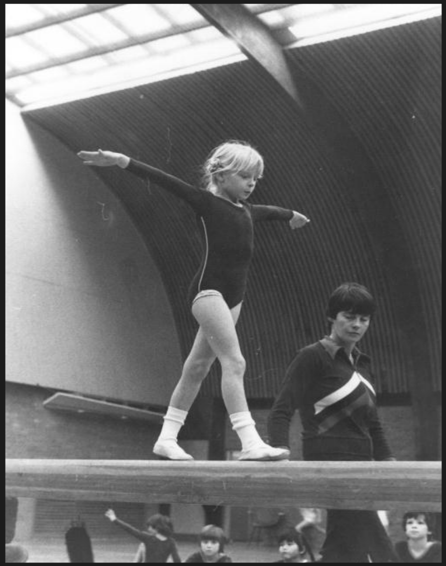
- regulation of Centre of Mass
  relative to base of support
  balancing
  correction steps

- labyrinths important for
  learning motor activities
  and fast feed back
  → automatisation




visual
cortex
cer
hippocampus
basal ganglia
spinal pattern generator
vn

otolith function especially relevant for:



motor learning (retardation in congenital areflexia)
maintaining complex postures
standing or slow walking
 on a soft surface (wind-surfing)
 in darkness
 in presence of misleading visual stimuli

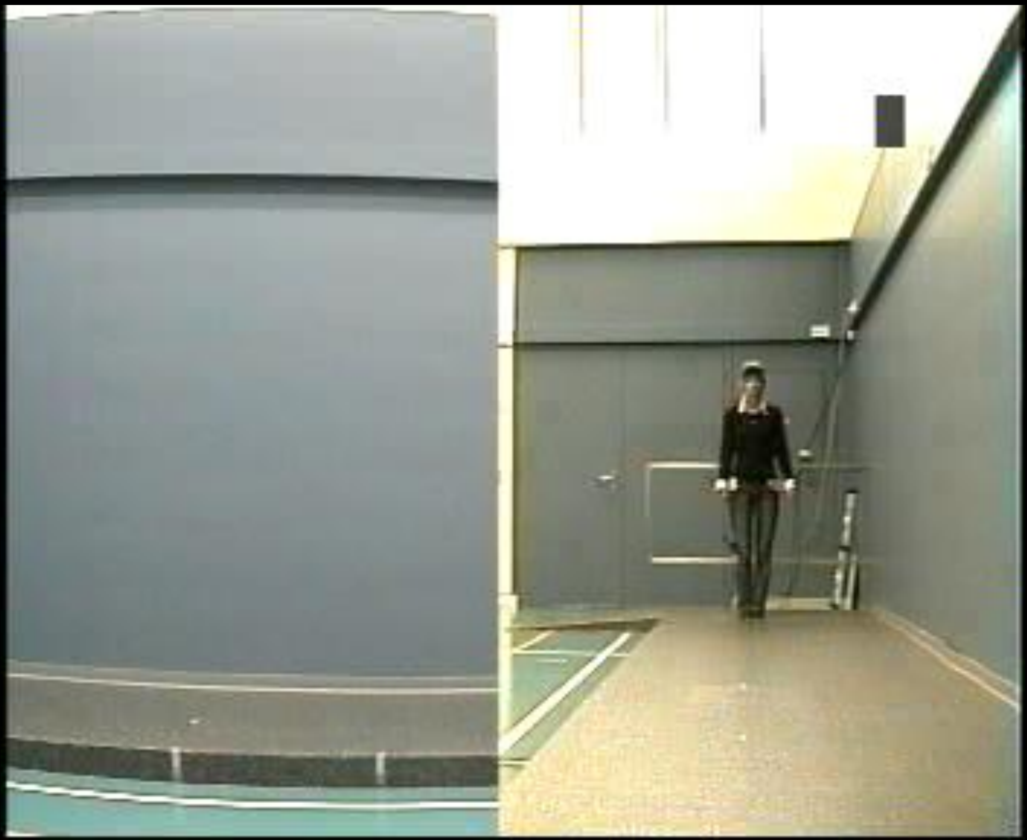
labyrinths less relevant for:

walking at normal speed or running (visual anticipation)

bilateral areflexia leads to degeneration of
“head direction” and head “place” cells in the hippocampus

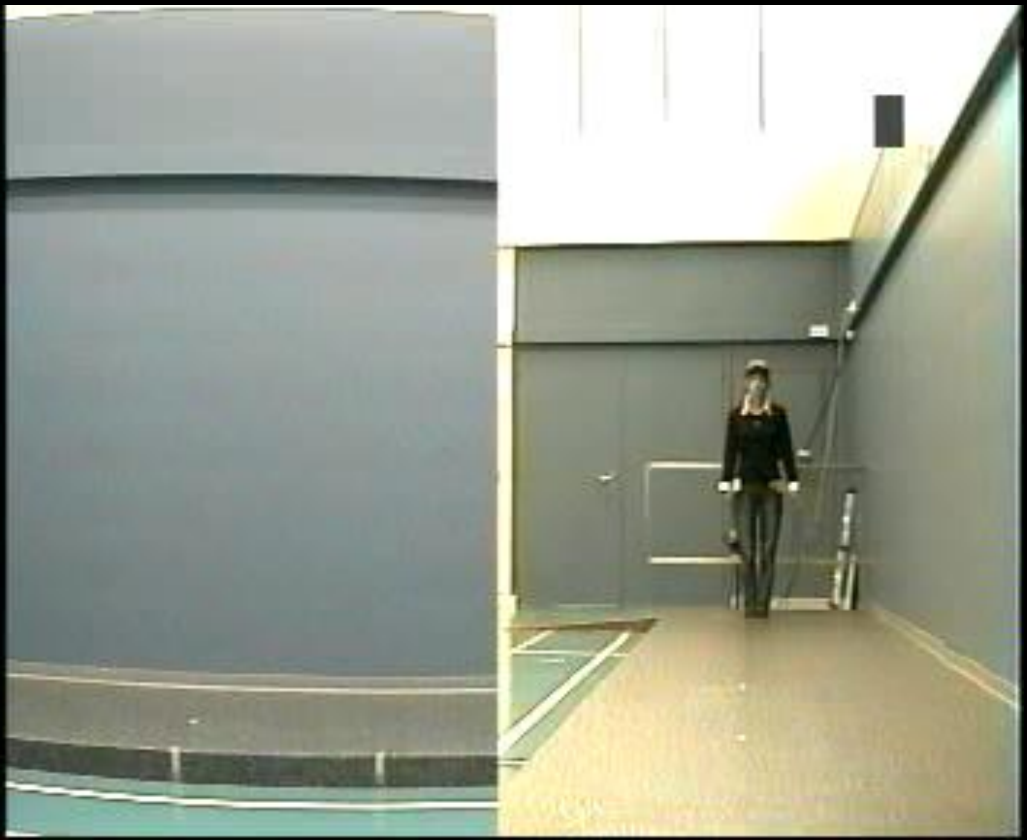
patient with severe bilateral vestibular hyporeflexia:

no more talking while walking (Brandt)



slow tandem walk

missing fast vestibular feed back



fast tandem walk

using visual anticipation

complaints related to vestibular dysfunction

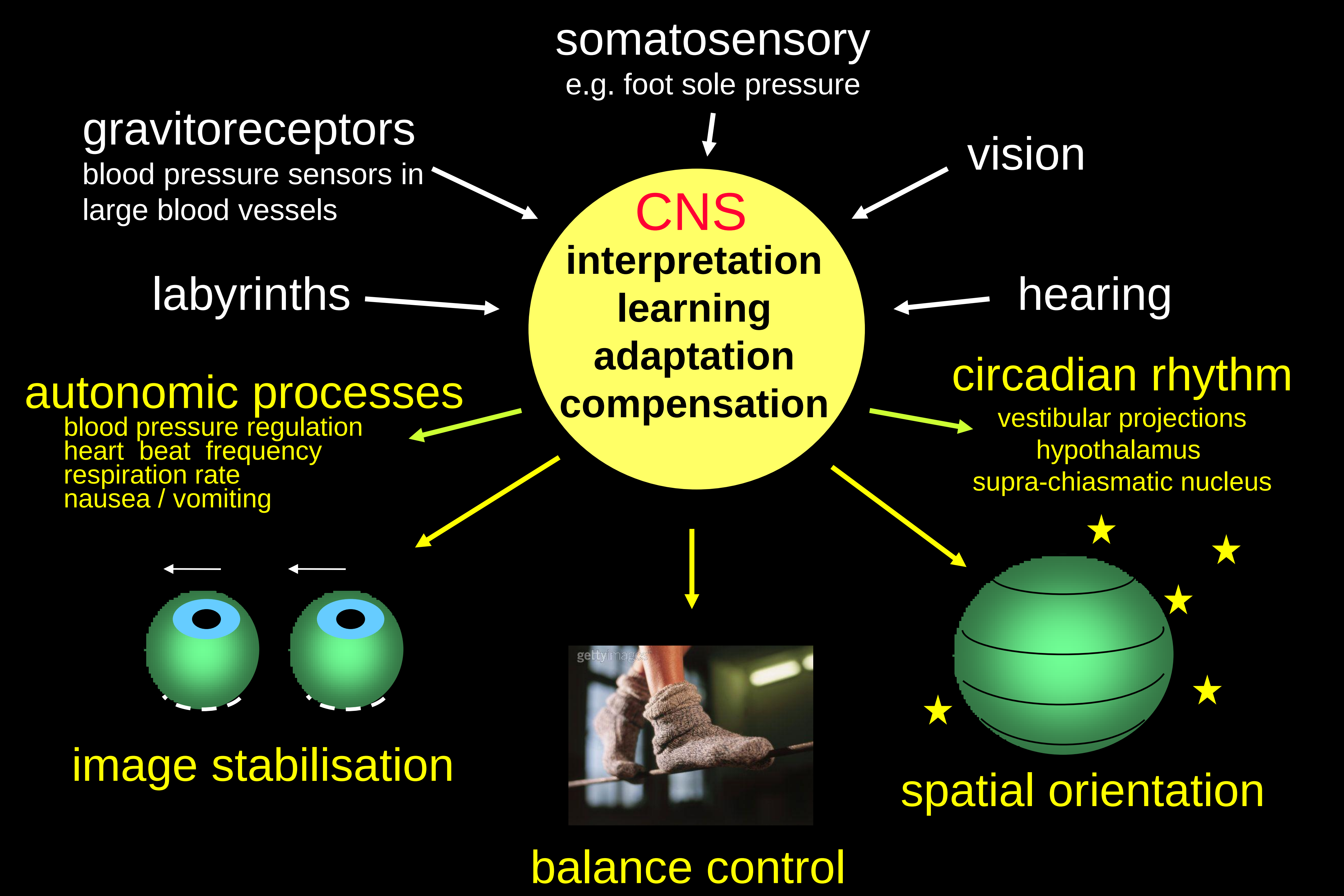
acute loss or fluctuating function

transient: vertigo, nausea, falling / imbalance

remaining peripheral vestibular function loss

sustained:

- enhanced neuro-vegetative sensitivity
- reduced ability to discriminate between self-motion and environmental motion
- reduced automatisation of balance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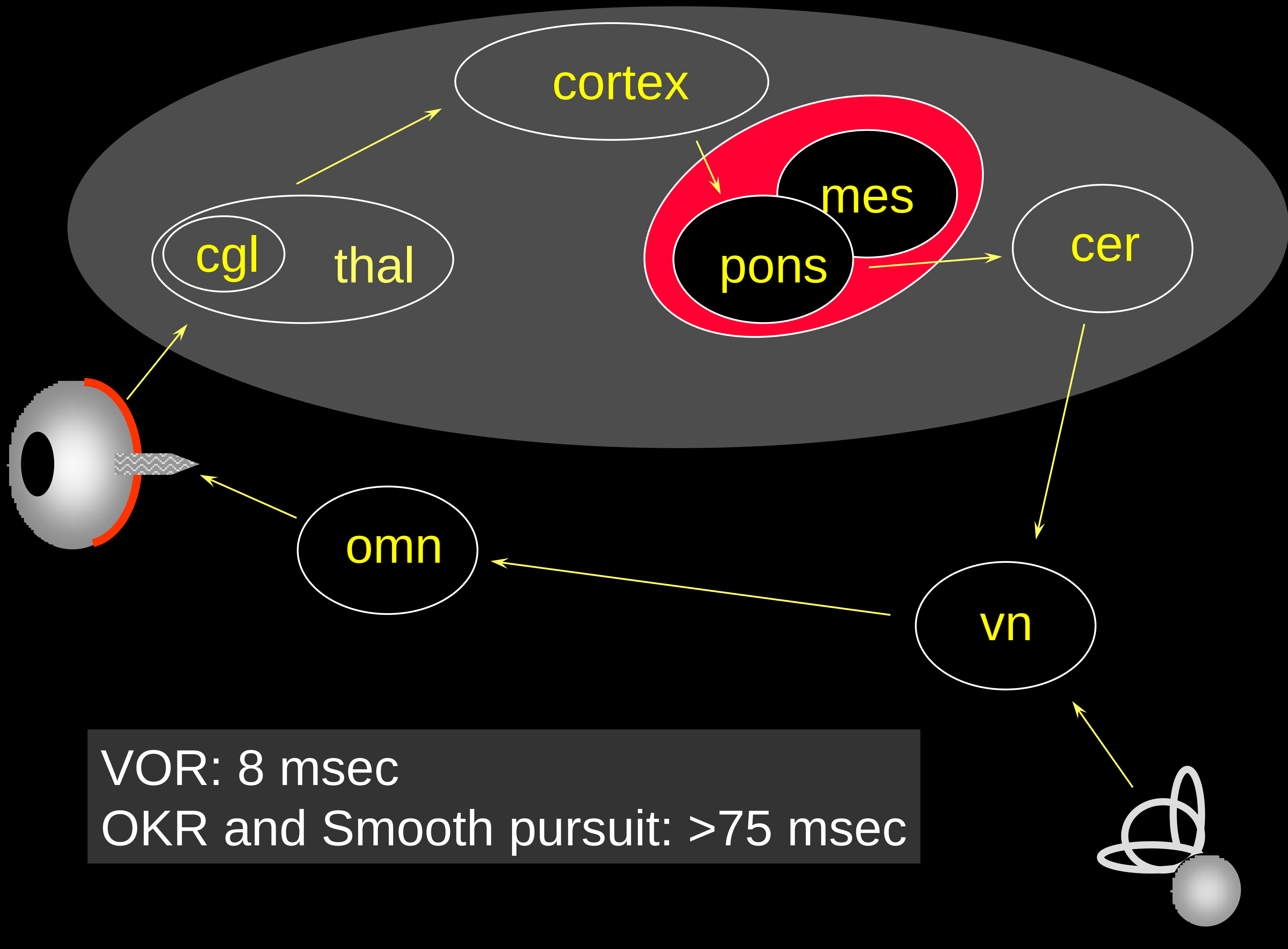

cortex
cgl
thal
mes
pons
cer
omn
vn
VOR: 8 msec
OKR and Smooth pursuit: >75 msec

head impulse test in unilateral loss

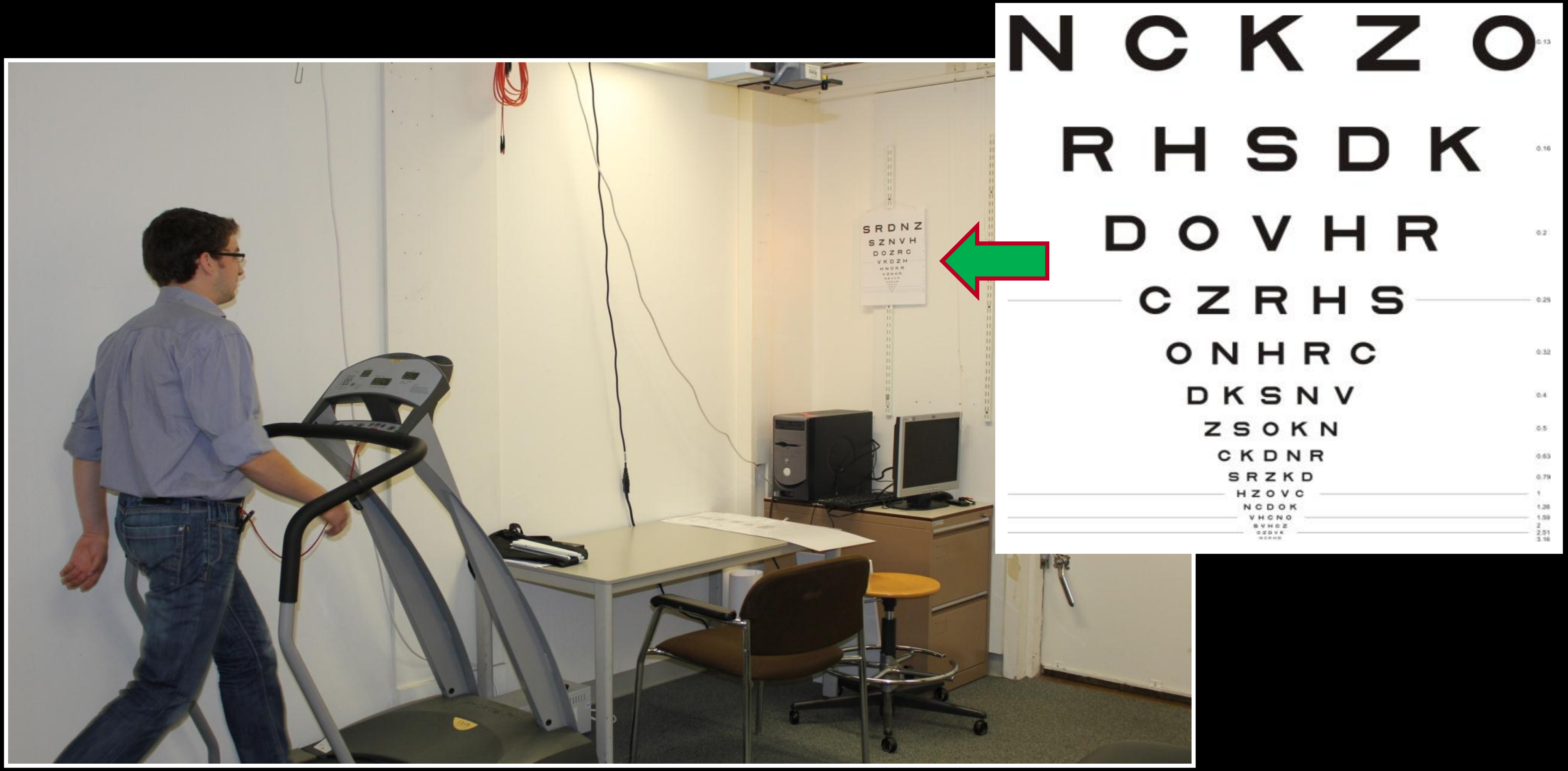
standard video (50 Hz)

pathology: central compensation

the other labyrinth does NOT take over

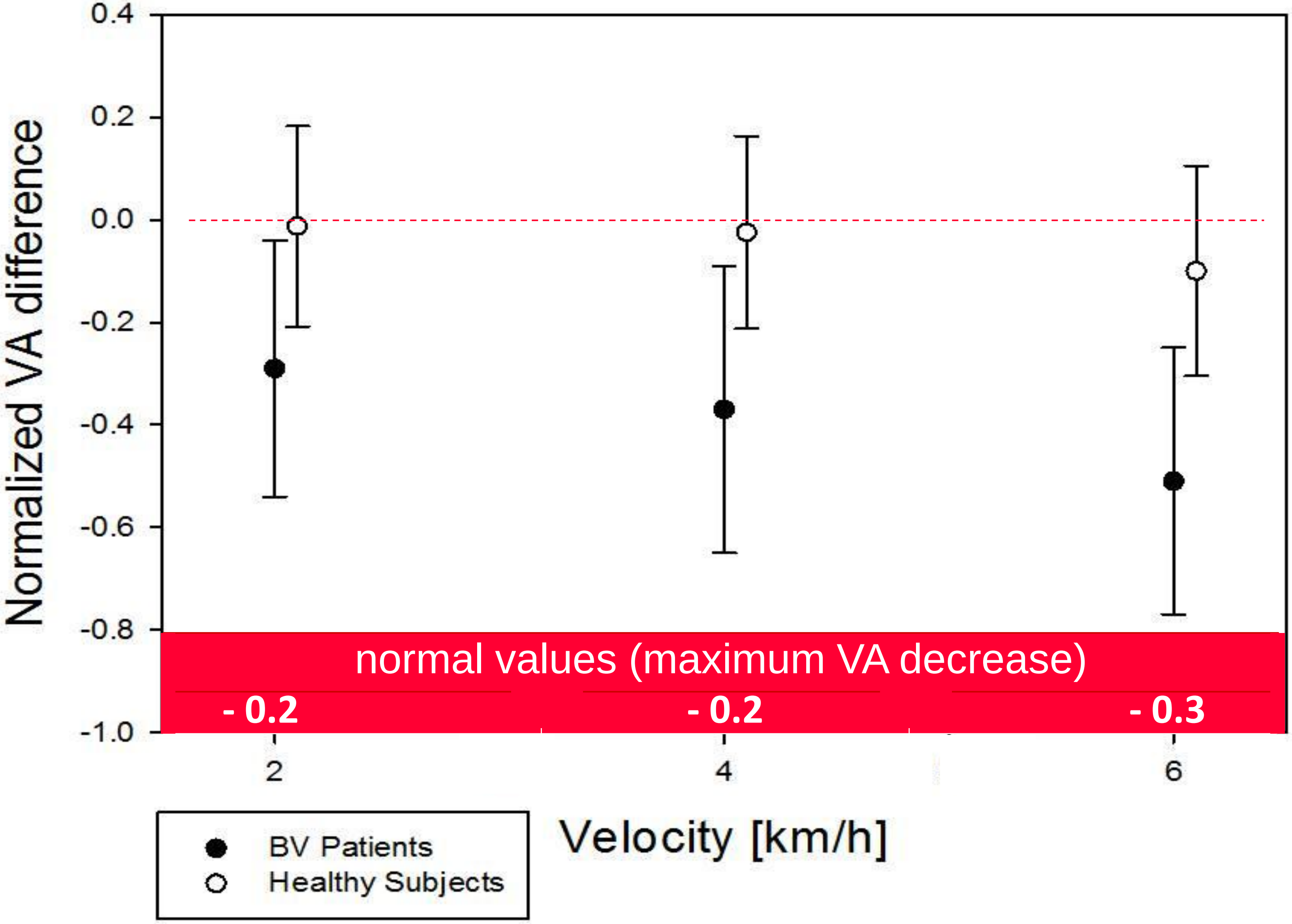
simulation of oscillopsia ≈ reduced dynamic visual acuity
in case of bilateral vestibular areflexia

Dynamic Visual Acuity (VA) measurement



treadmill: 2, 4 and 6 km/h

decrease of VA during walking



which complaints are related to vestibular deficits ?

which complaints are related to natural limitations ?

acute unilateral:

- vertigo, imbalance, nystagmus

sustained:

- impact on various autonomic functions
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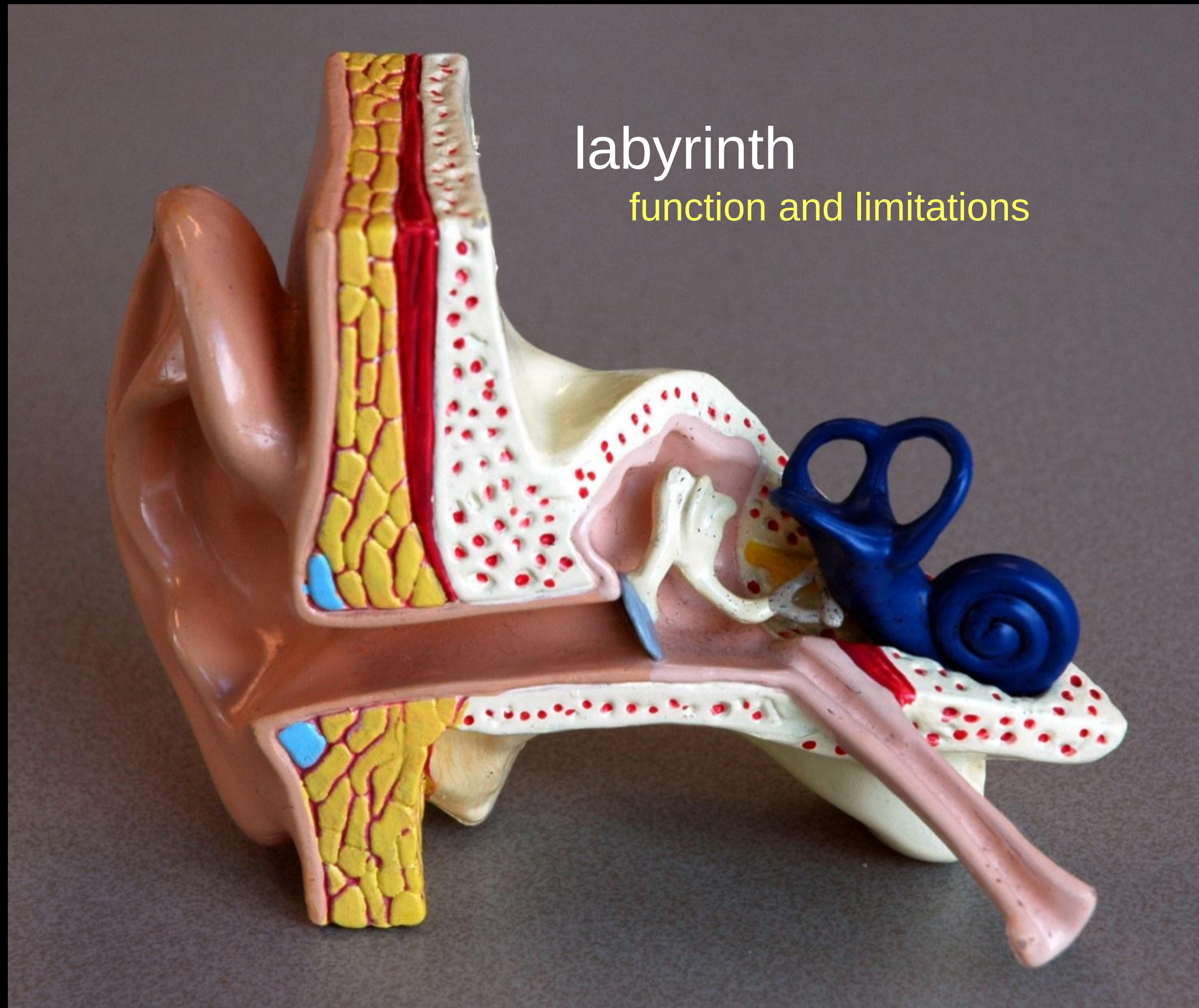
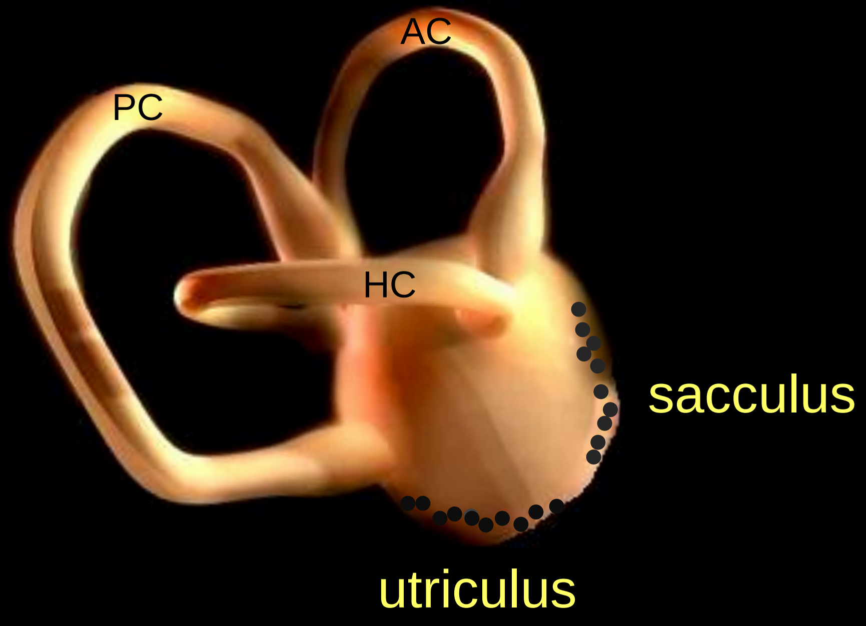
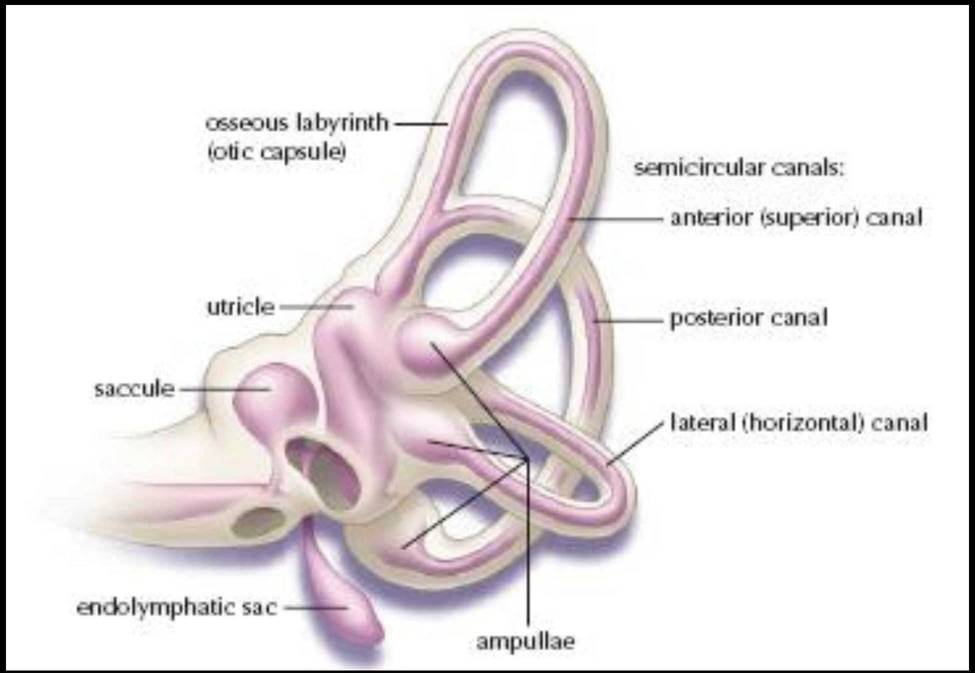
labyrinth

function and limitations



vestibular labyrinth senses low frequency motions: movement
cochlear labyrinth senses high frequency motions: sound

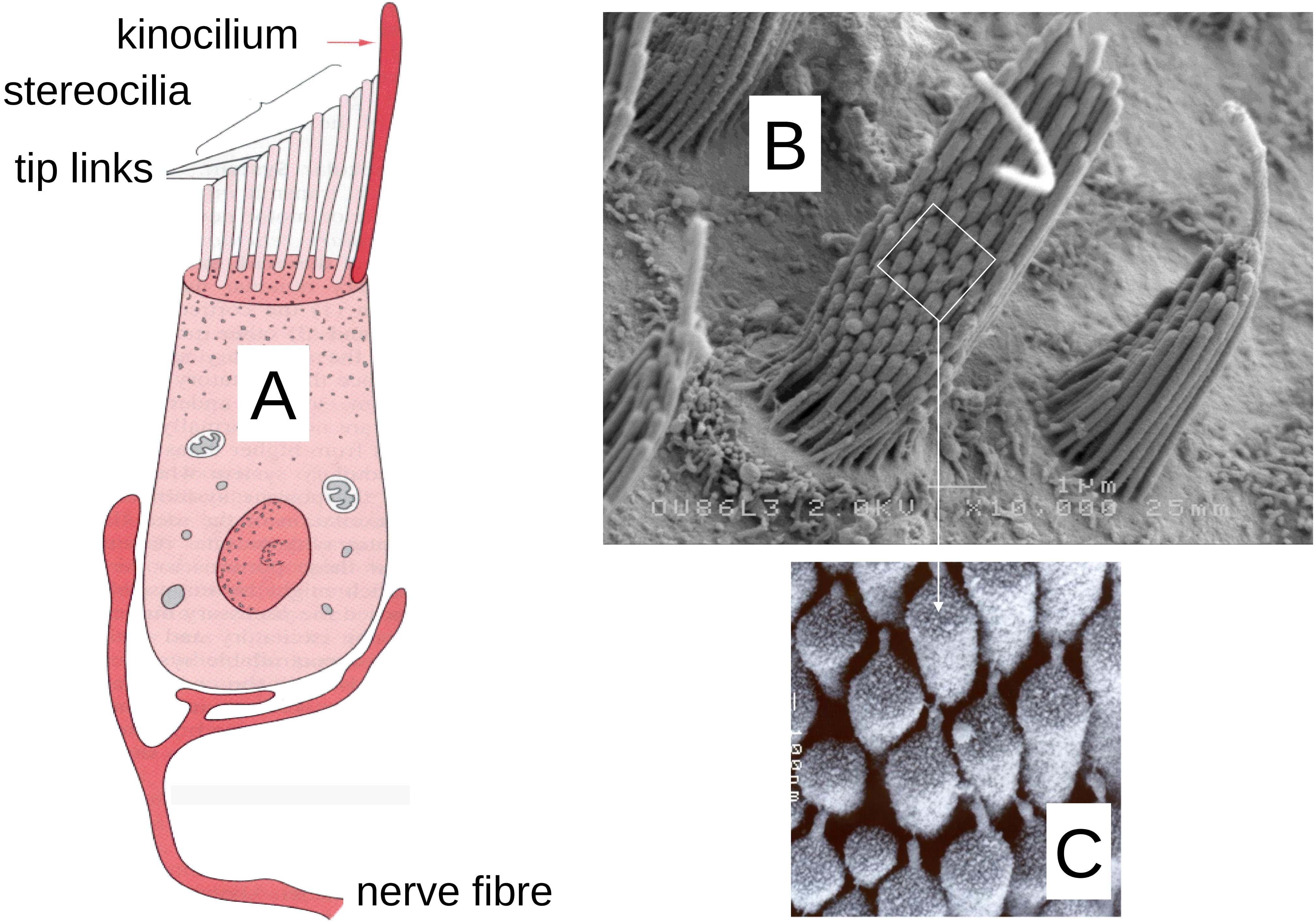




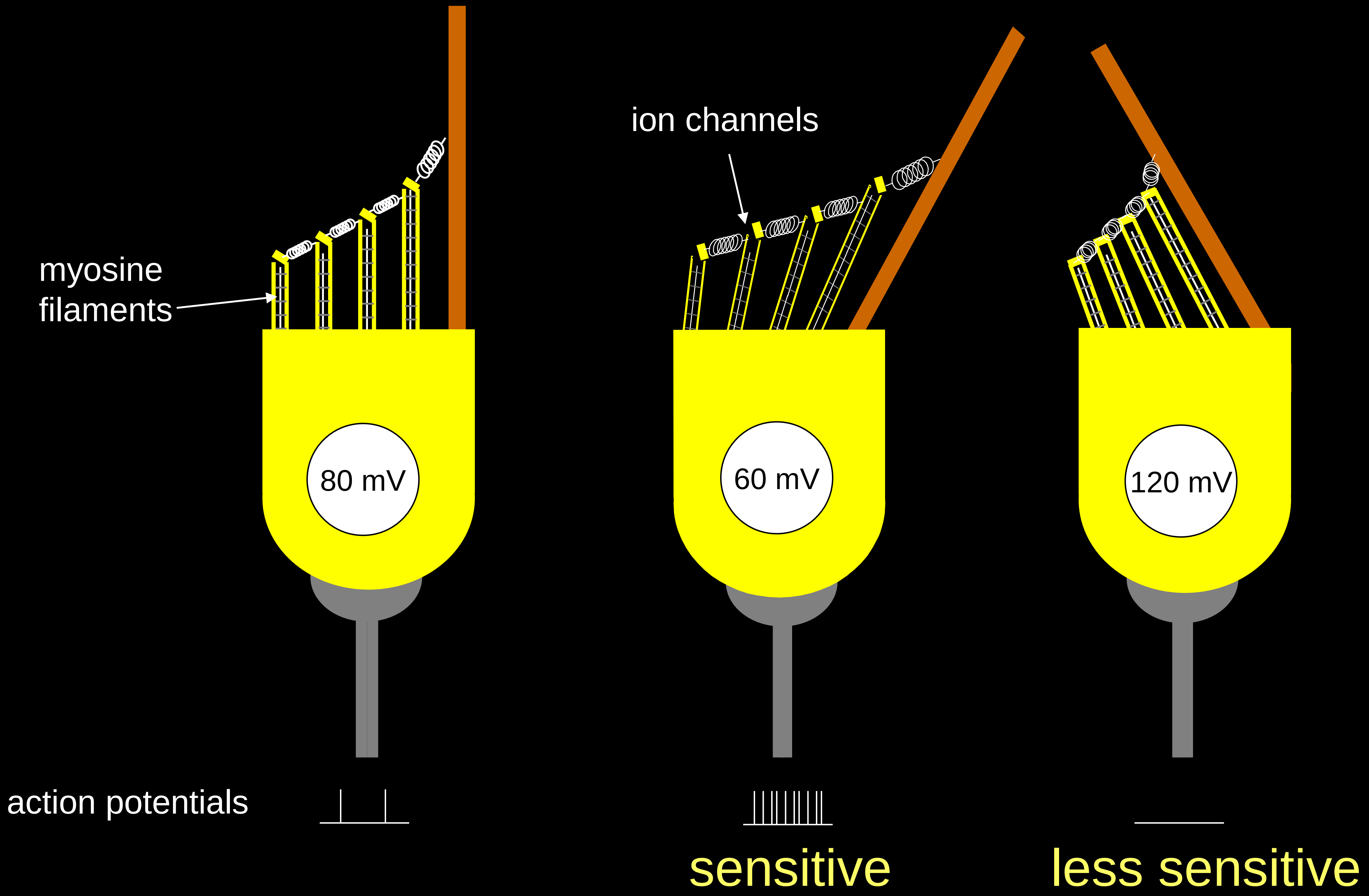
vestibular labyrinth senses head movement and tilt

rotations: 3 canals HC+PC+AC

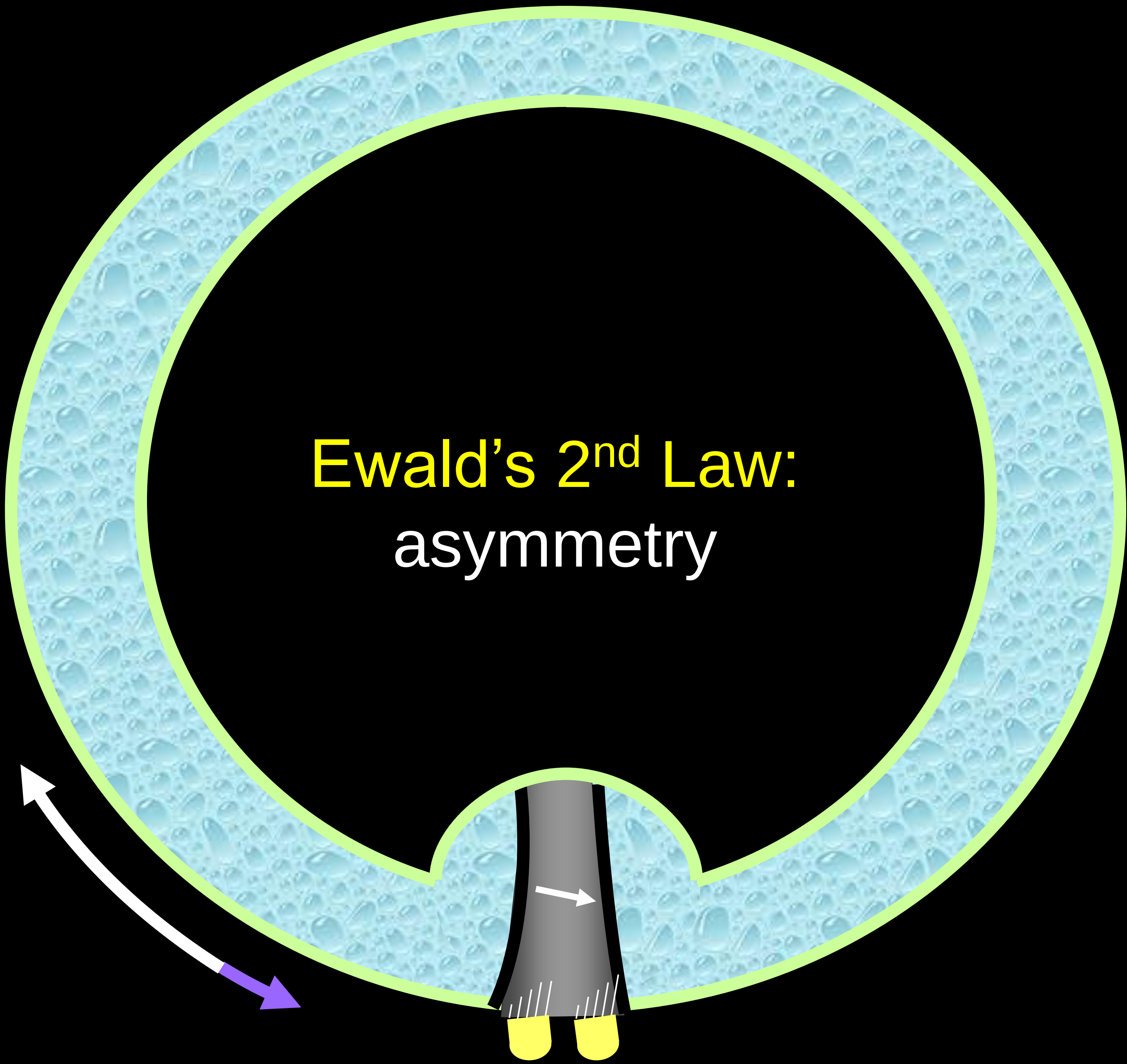
translations + tilt: statolith (utriculus + sacculus)


kinocilium
stereocilia
tip links
A
nerve fibre
B
C

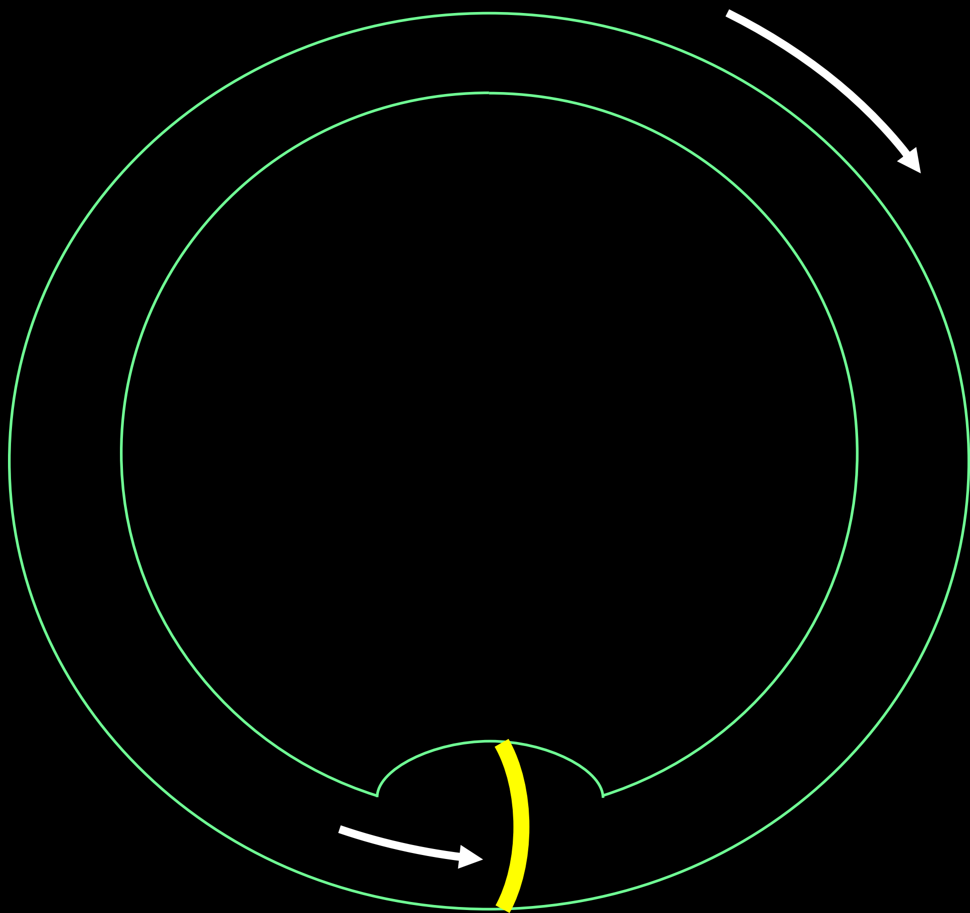
Ewald's 2nd Law: asymmetry



Ewald's 2nd Law: asymmetry



acceleration / inertia of mass → ← elasticity viscosity (friction)



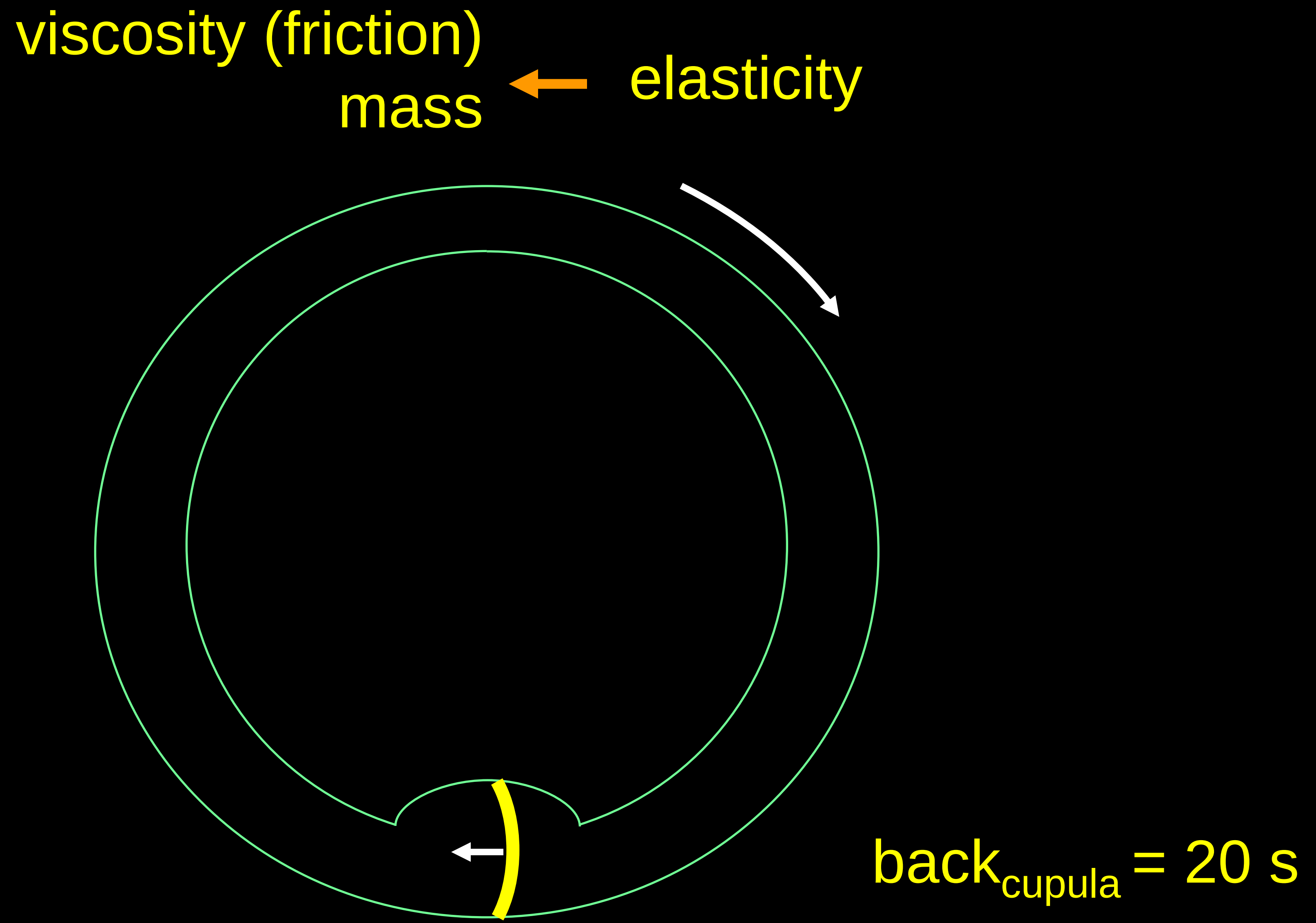
latency SP = 0.8 ms

max. deflection$_{cupula}$ = 2 ms

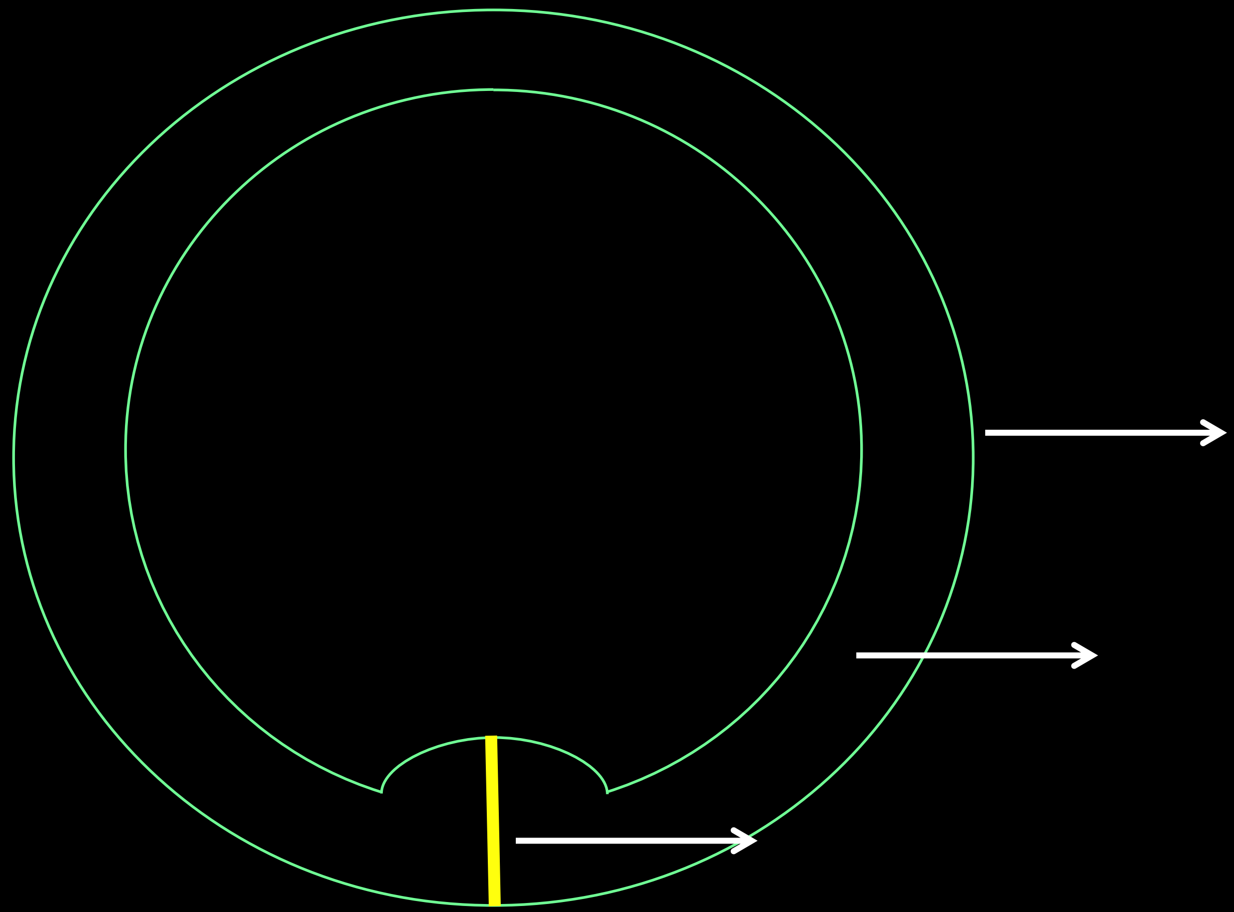
latency VOR = 8 ms

maximum deflection ≈ 1°

canals are insensitive for constant rotations

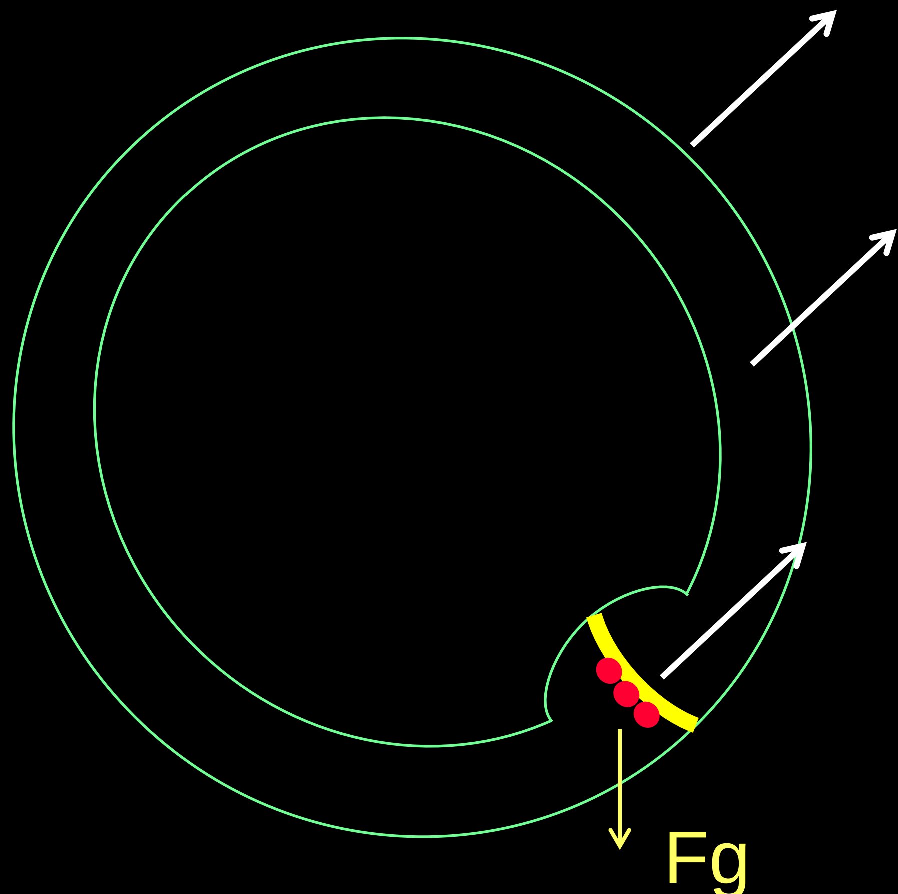


canals are insensitive for translations or gravity
(specific mass endolymphe = specific mass cupula)



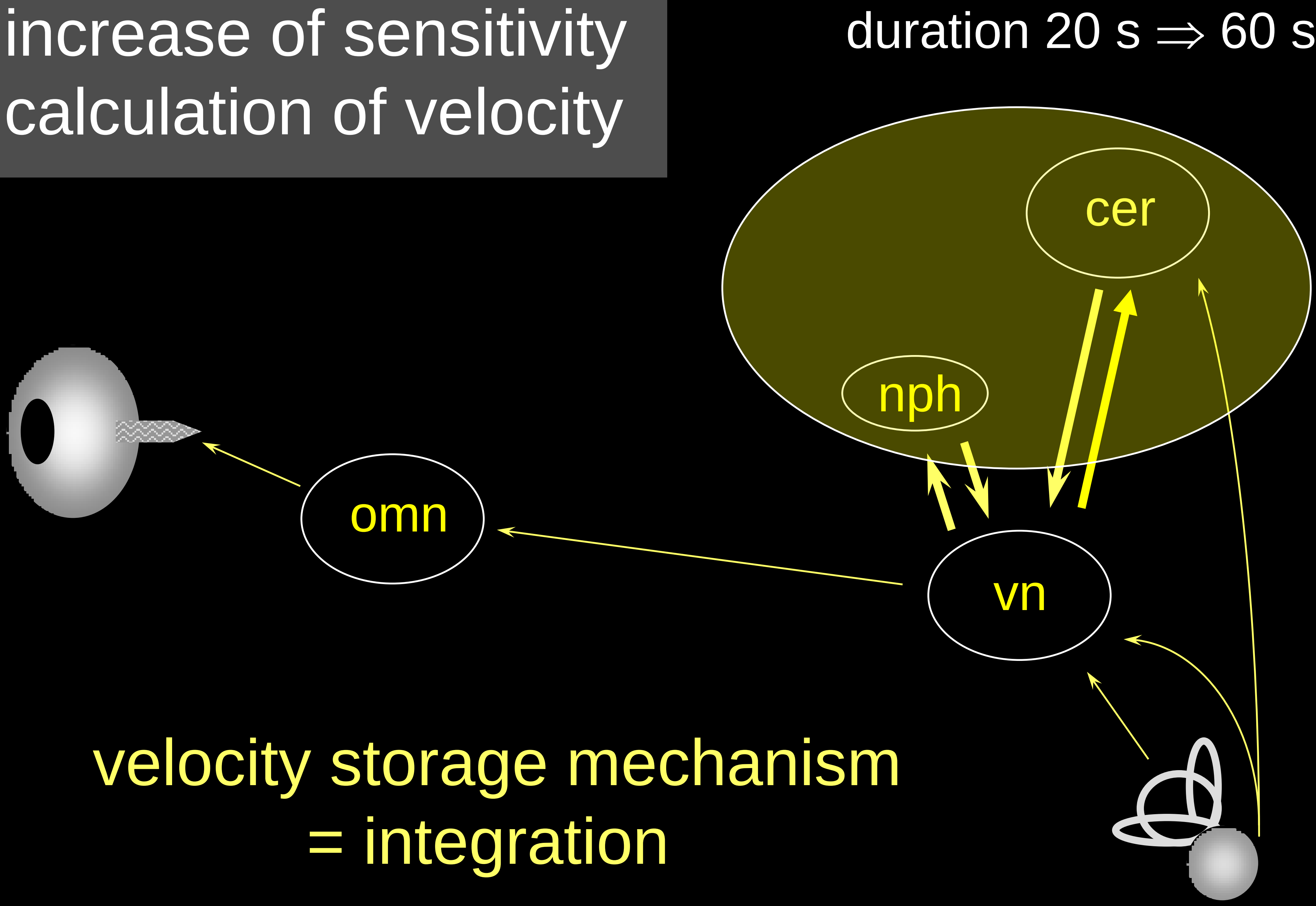
exceptions: alcohol, cupulolithiasis etc canals are insensitive for translations or gravity

(specific mass endolymphe = specific mass cupula)

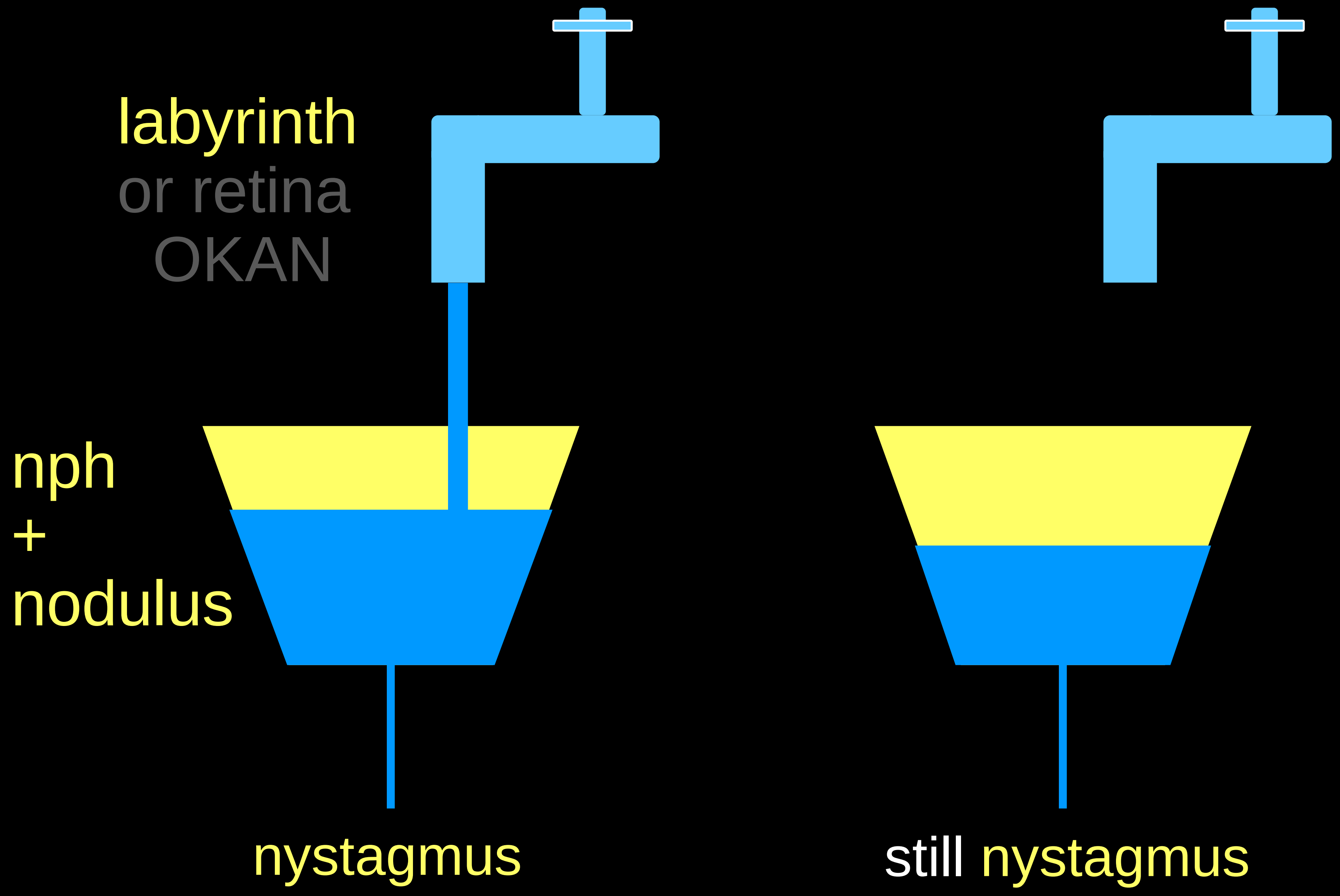


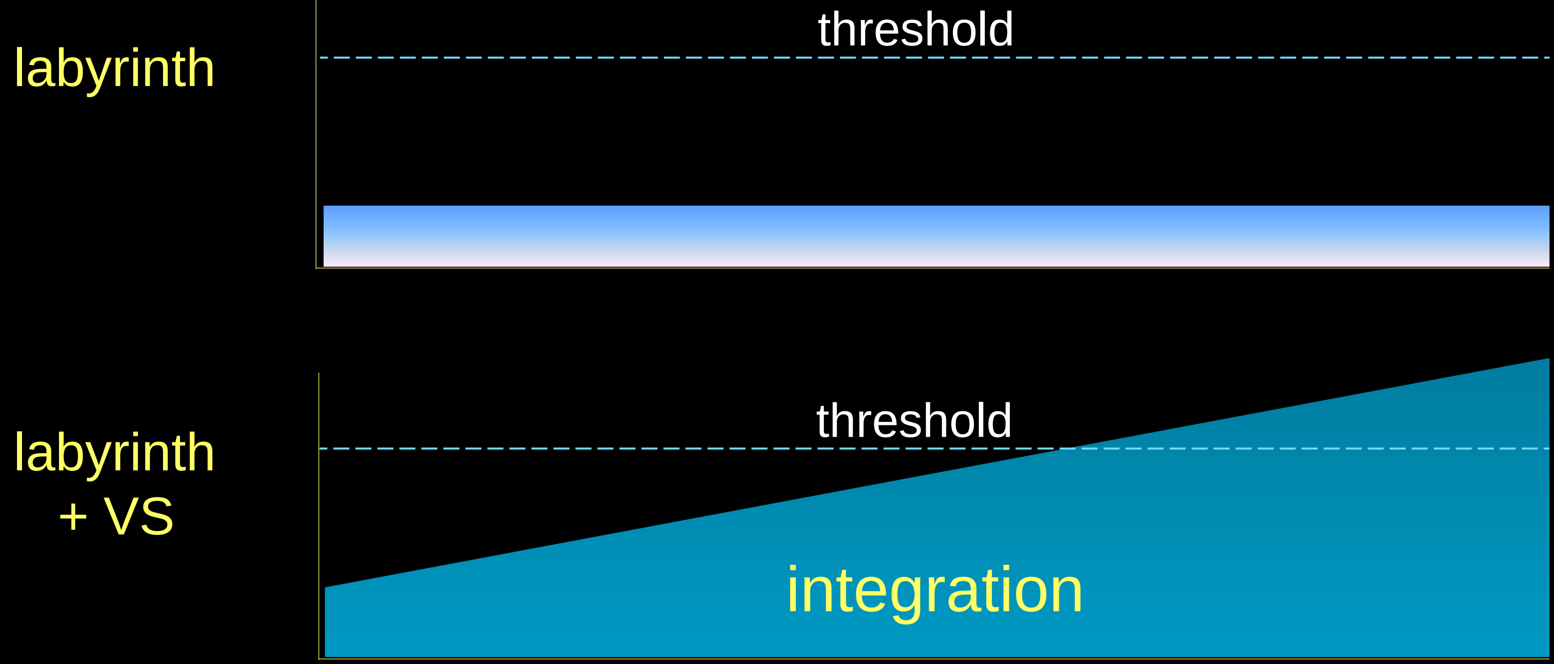
exceptions: alcohol, cupulolithiasis etc

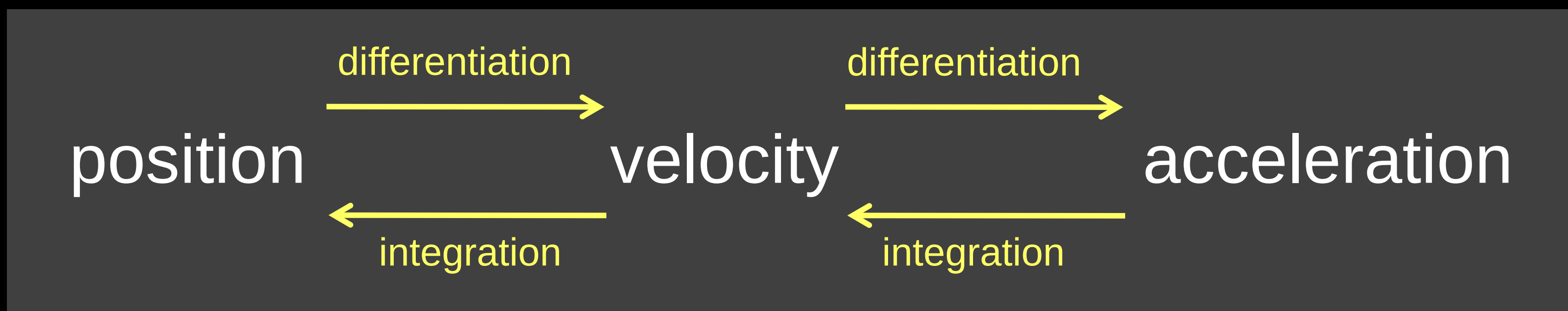
- increase of sensitivity
- calculation of velocity



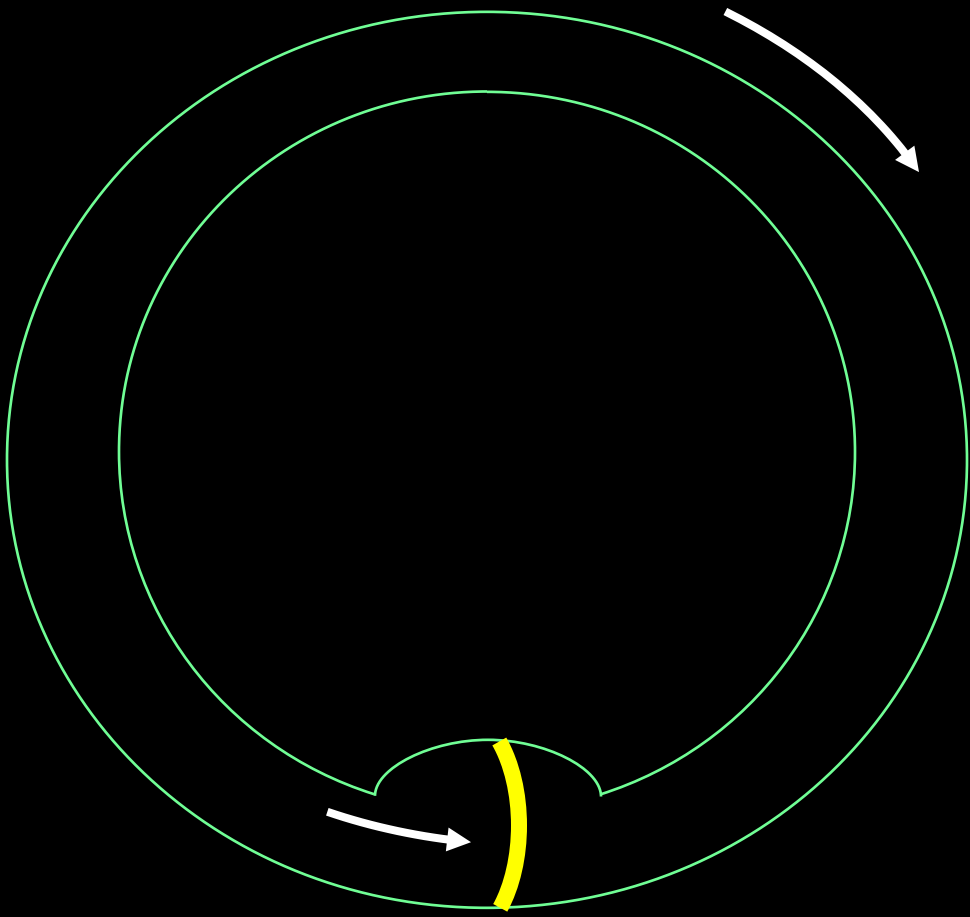
velocity storage mechanism
= integration

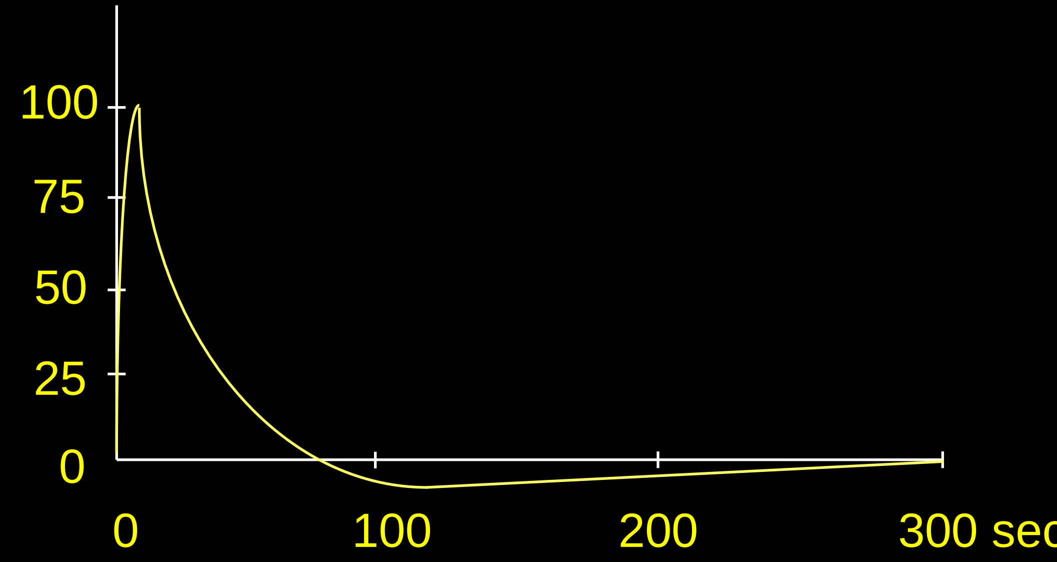

labyrinth
or retina
OKAN
nph
+
nodulus
nystagmus
still nystagmus





- canal detects head acceleration
- brain calculates head velocity
- brain matches head and eye velocity = SPV





$\text{duration}_{\text{deflection cupula}}$ = 2 ms

$\text{duration}_{\text{cupula back}}$ = 20 s

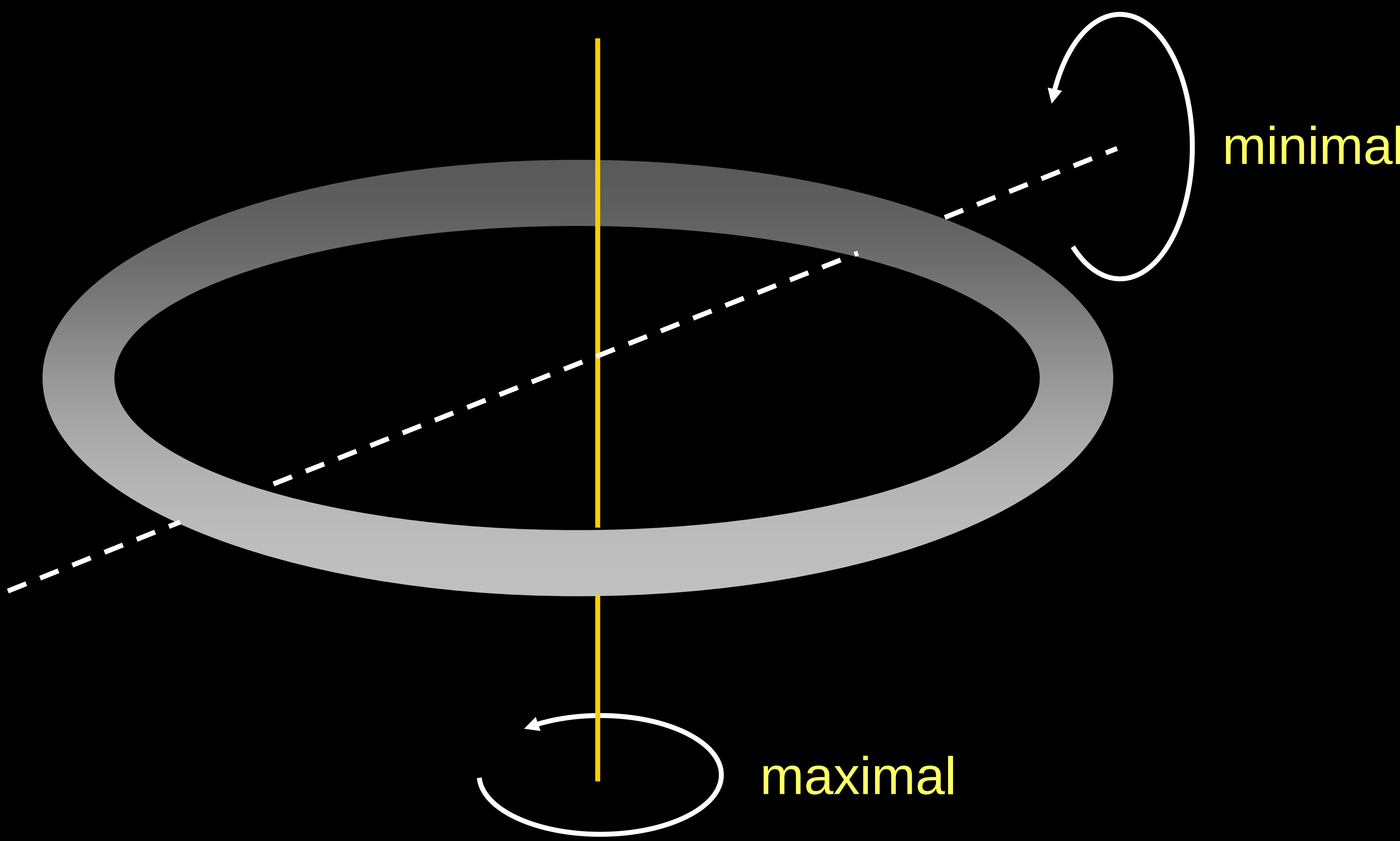
$\text{duration}_{\text{velocity storage}}$ = 60 s

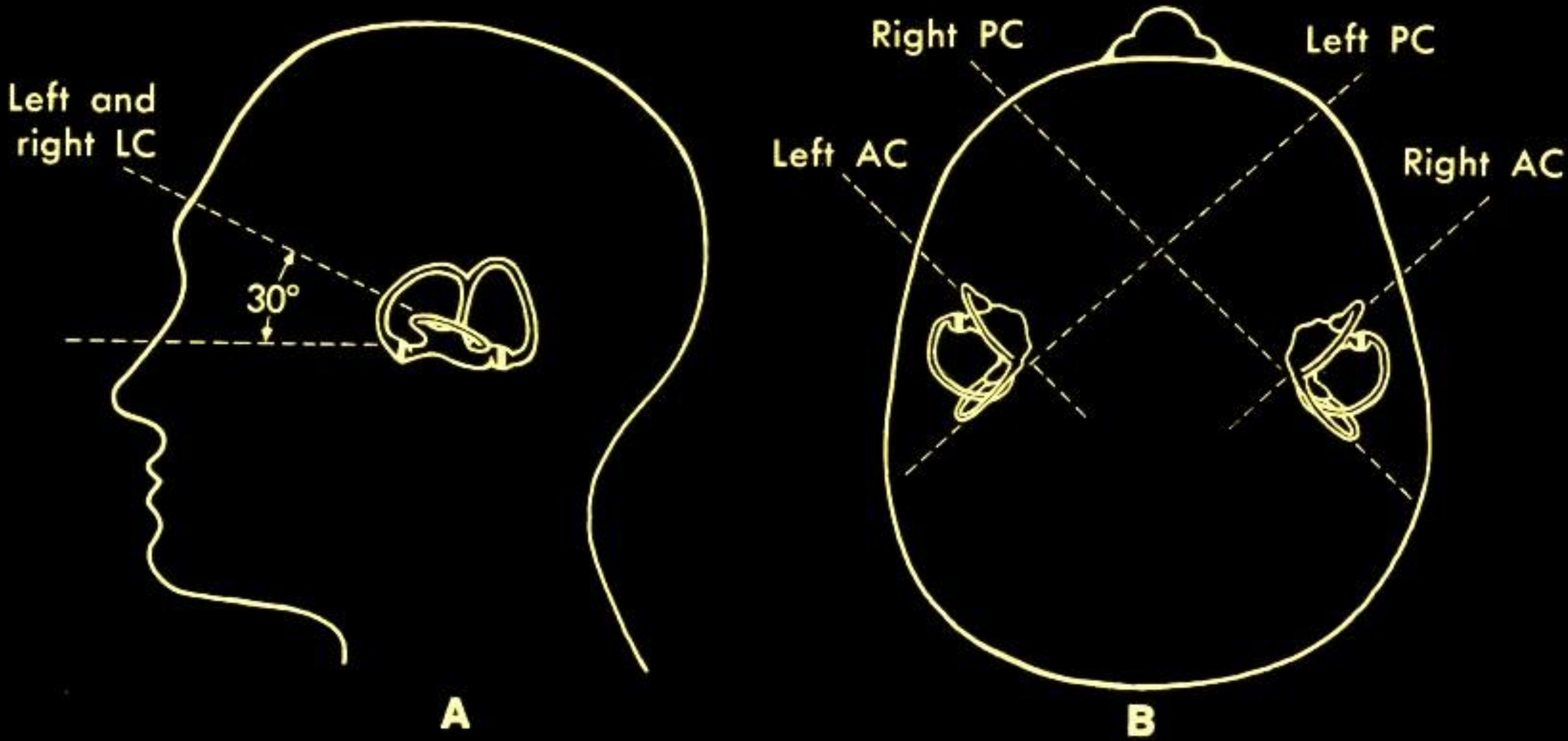
$\text{duration}_{\text{central adaptation}}$ > 300 s

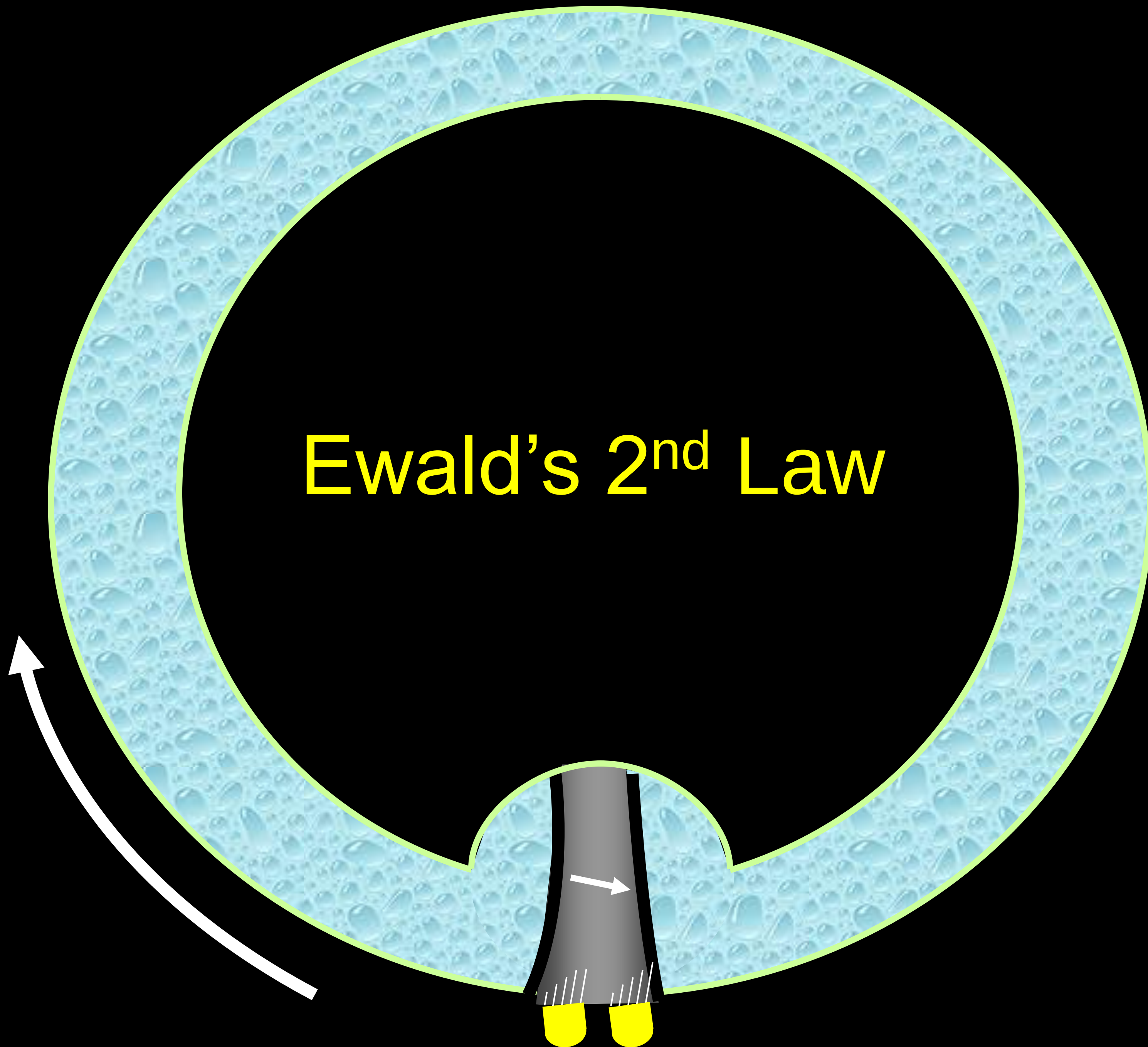
velocity storage: mainly for horizontal canals

Ewald's 1st Law: optimal sensitivity

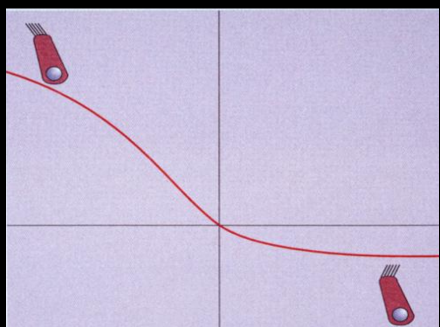
we need 3 dimensions: 3 canals

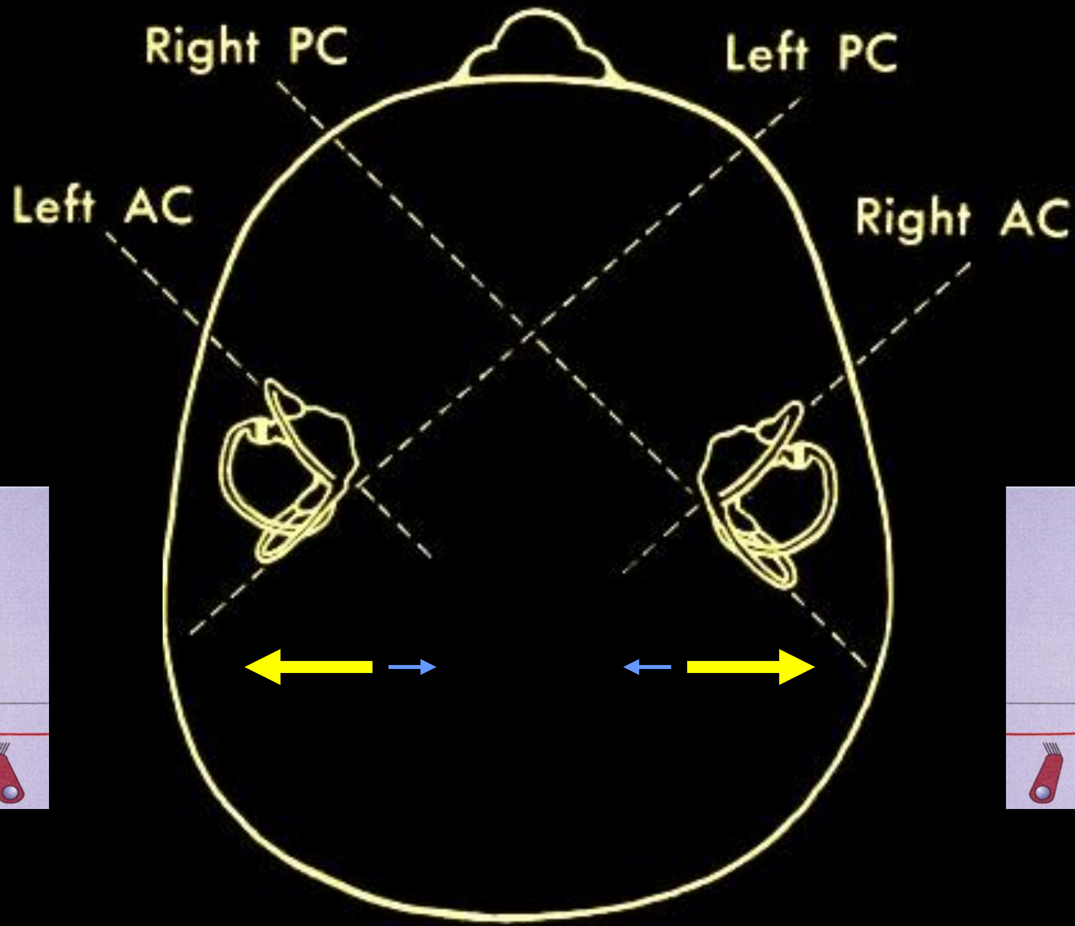


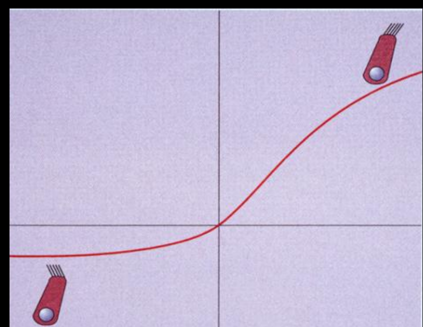

Left and
right LC
30°
A
Right PC
Left PC
Left AC
Right AC
B


Ewald's 2nd Law

The German Experience







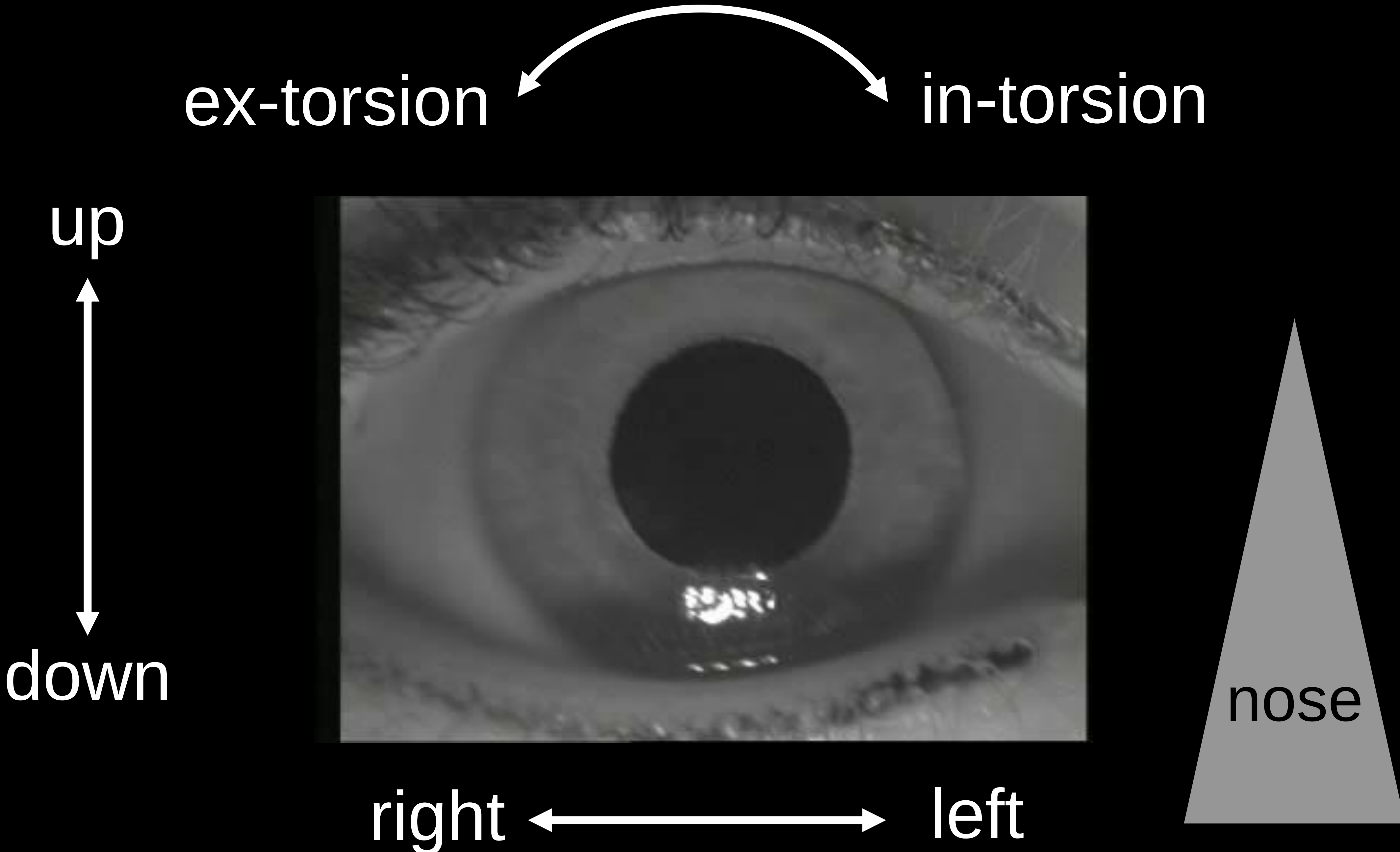
loss of gaze stabilisation (towards bad-side)
especially for fast head movements

VOR 3D: nystagmus 3D

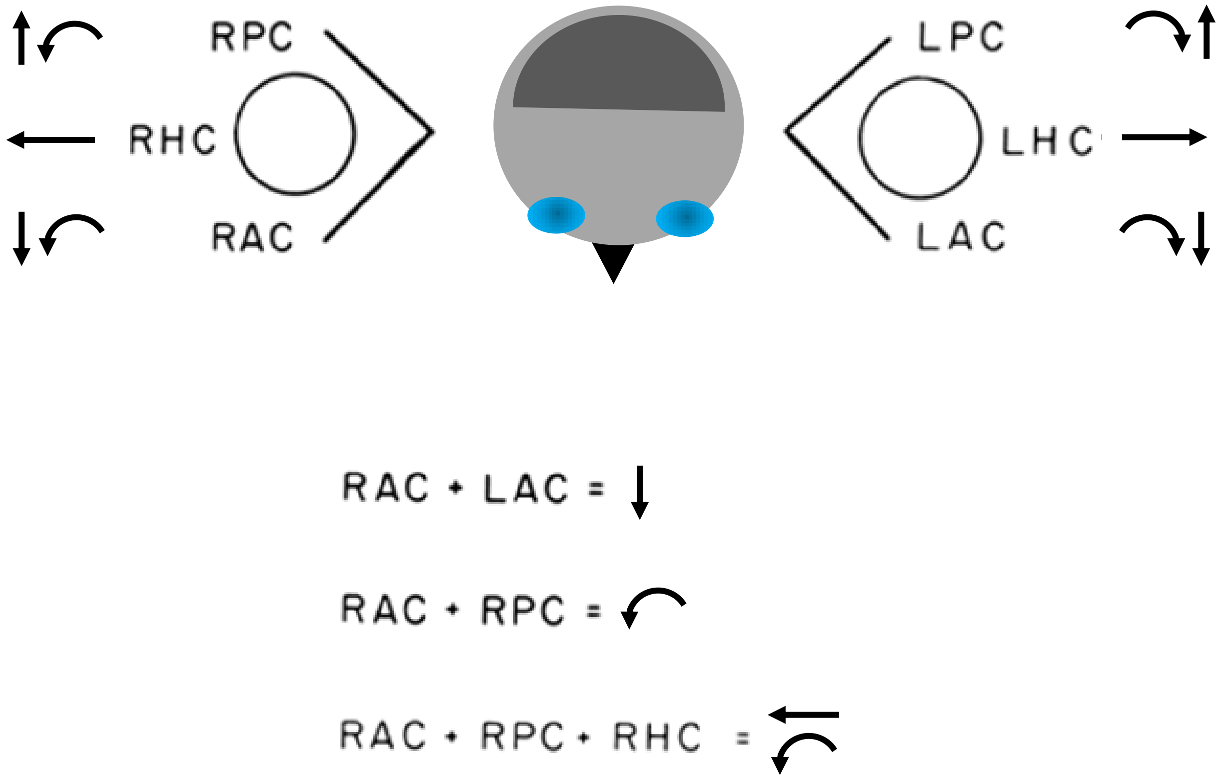
direction = fast phase

magnitude = slow phase

horizontal (left – right)
vertical (up – down)
torsional (in- and extorsion)


ex-torsion
in-torsion
up
down
nose
right
left

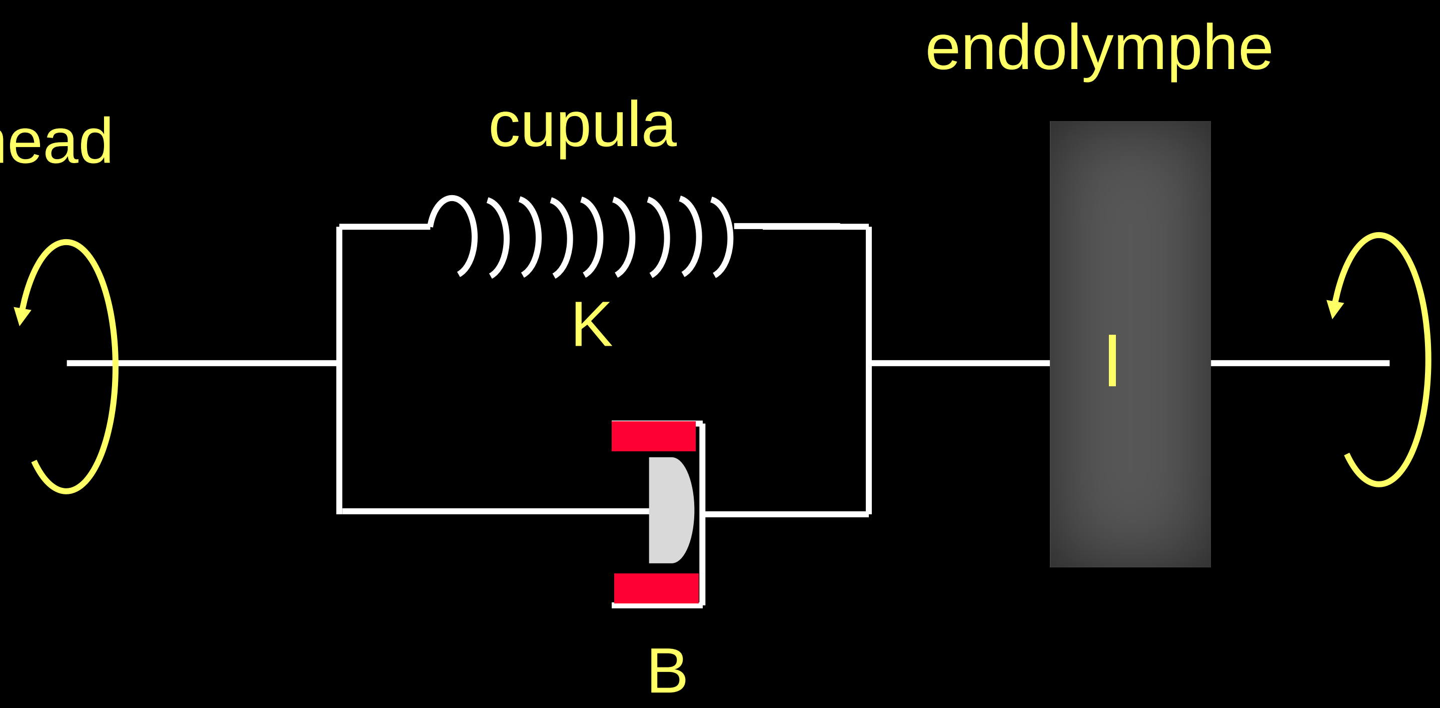
direction nystagmus **FAST** phase induced by stimulation



vertical-rotatory or horizontal-rotatory: peripheral
horizontal: peripheral or central
pure vertical or pure rotatory: central

frequency dependence
semicircular canals ?

cupula deflection depends on viscosity, elasticity and mass



theoretical model canal: 2nd order system

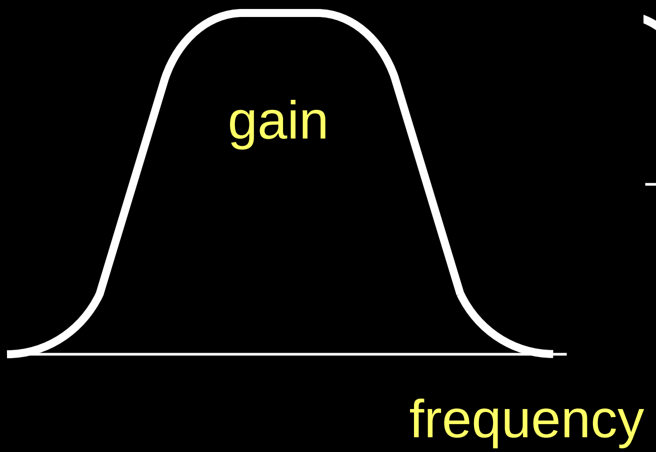
B ~ friction / viscosity (max. friction: endolymphe moves with canal)
K ~ elasticity cupula (no elasticity: cupula does not bend back)
I ~ endolymphe mass, size (no inertia: no movement)

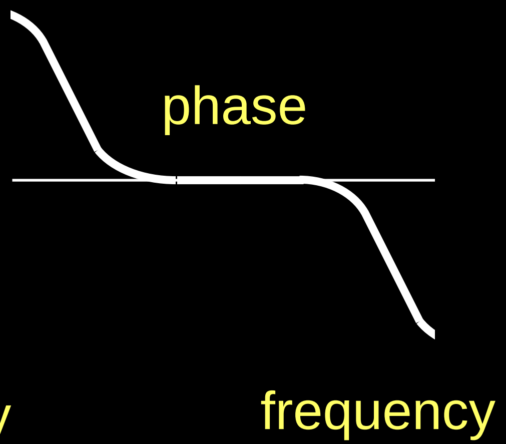
cupula deflection depends on viscosity, elasticity and mass

theoretical model canal: 2nd order system
leads to the following differential equation

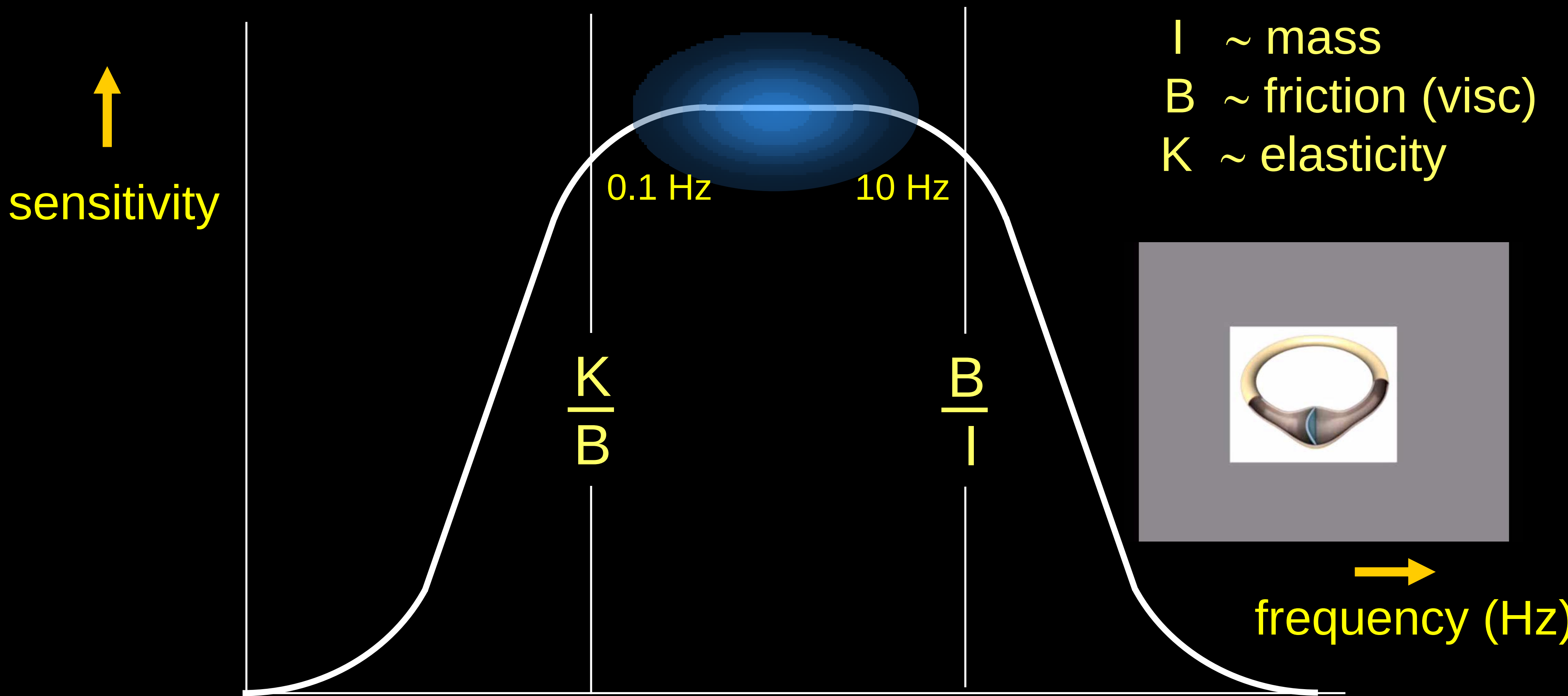
$$\ddot{q} = \ddot{\Theta} + \frac{B}{I}\dot{\Theta} + \frac{K}{I}\Theta$$

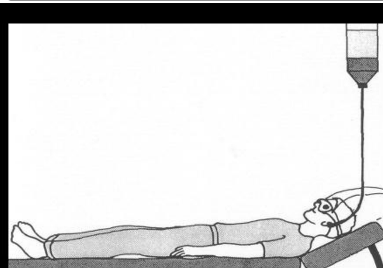
q angle head rotation
Θ angle cupula deflection
I ~ endolymphe mass, size
B ~ friction (viscosity)
K ~ elasticity cupula





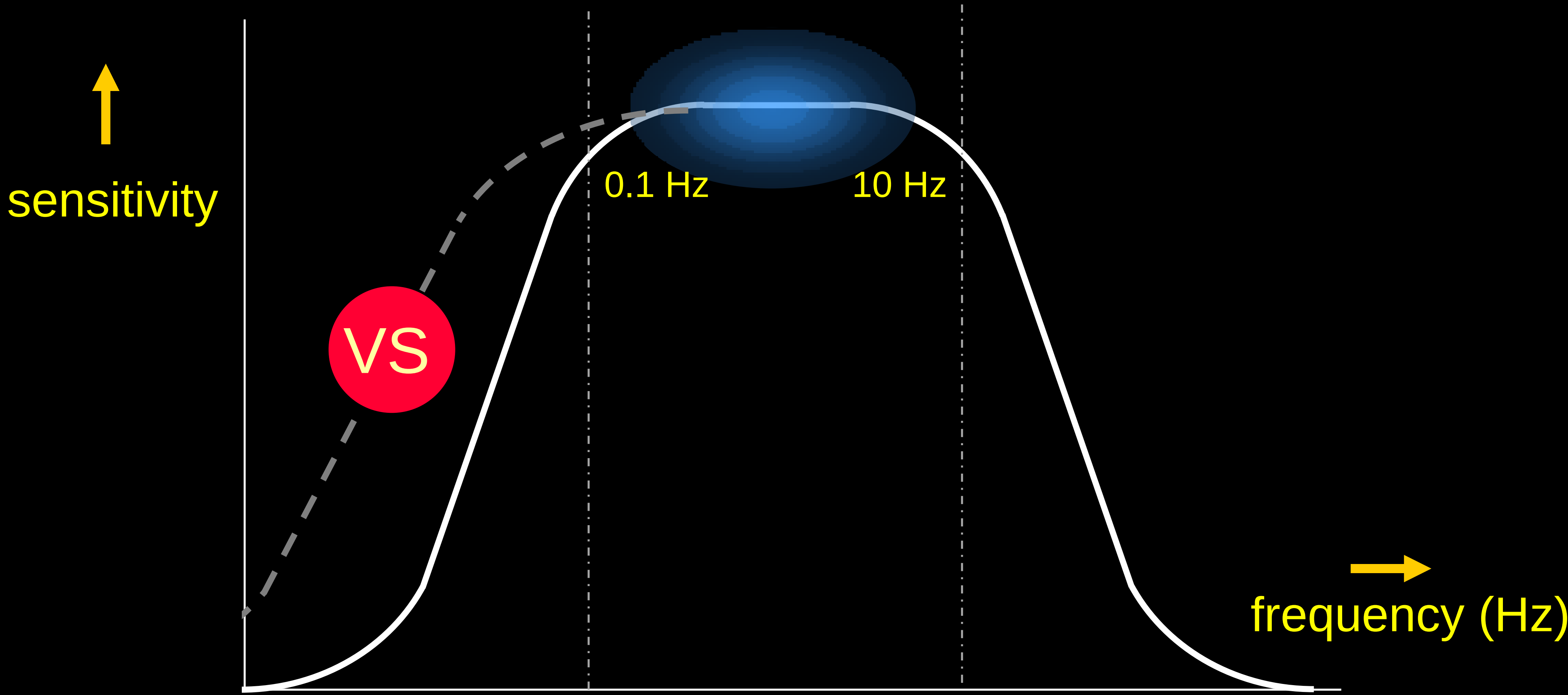
frequency dependence canals: gain



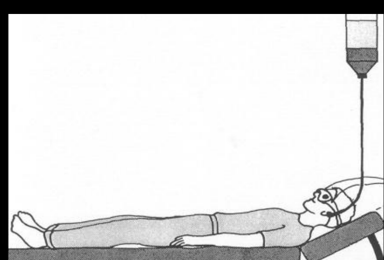


canal senses acceleration, cupula deflection indicates head velocity

frequency dependence canals: gain

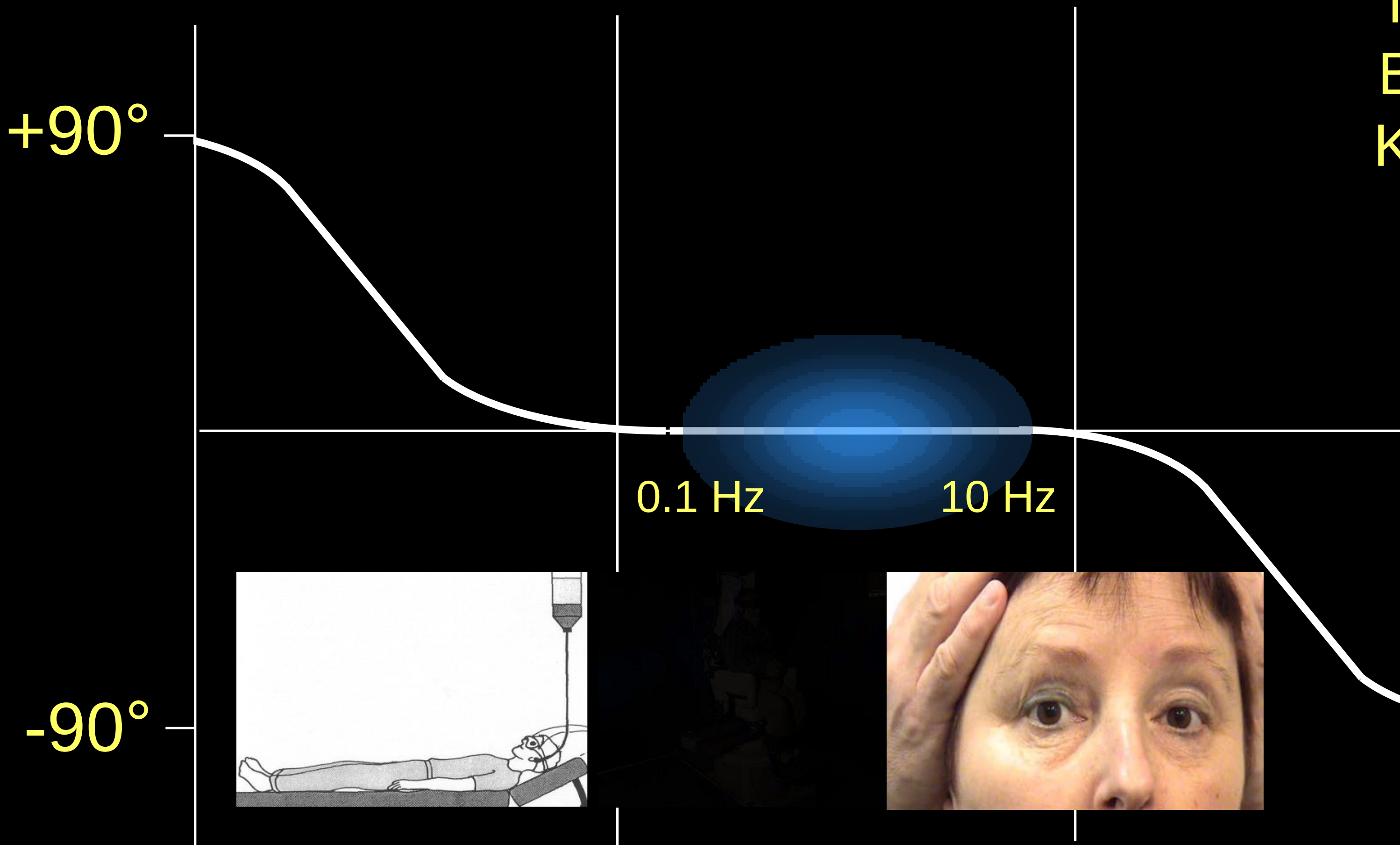


calorics chair head impulses



frequency dependence canals: phase

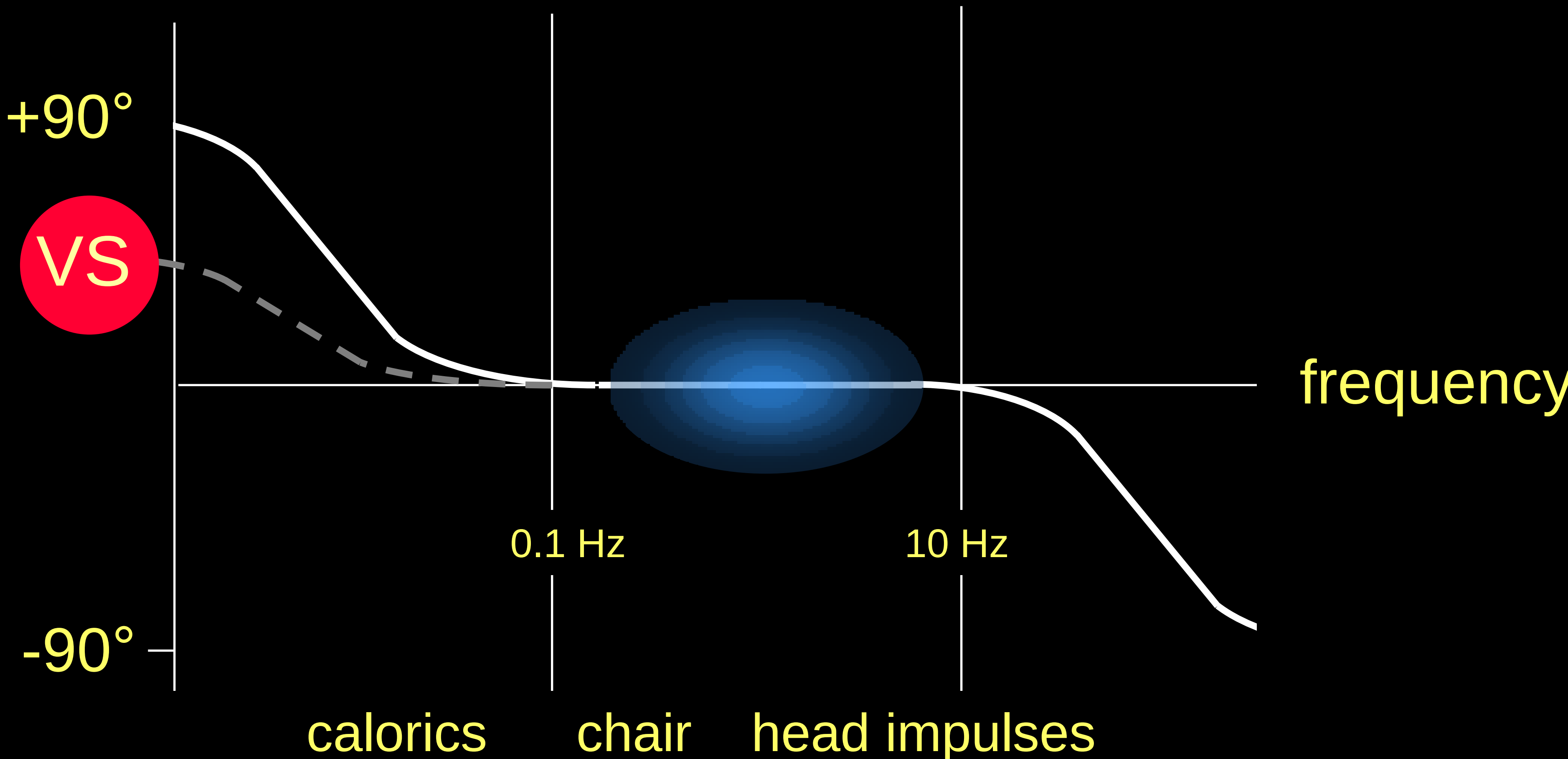
I ~ mass
B ~ friction (visc)
K ~ elasticity

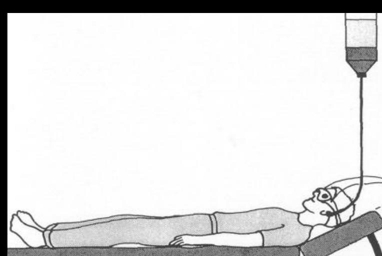


$1 / T_{low} = \dfrac{K}{B}$ $1 / T_{high} = \dfrac{B}{I}$ frequency

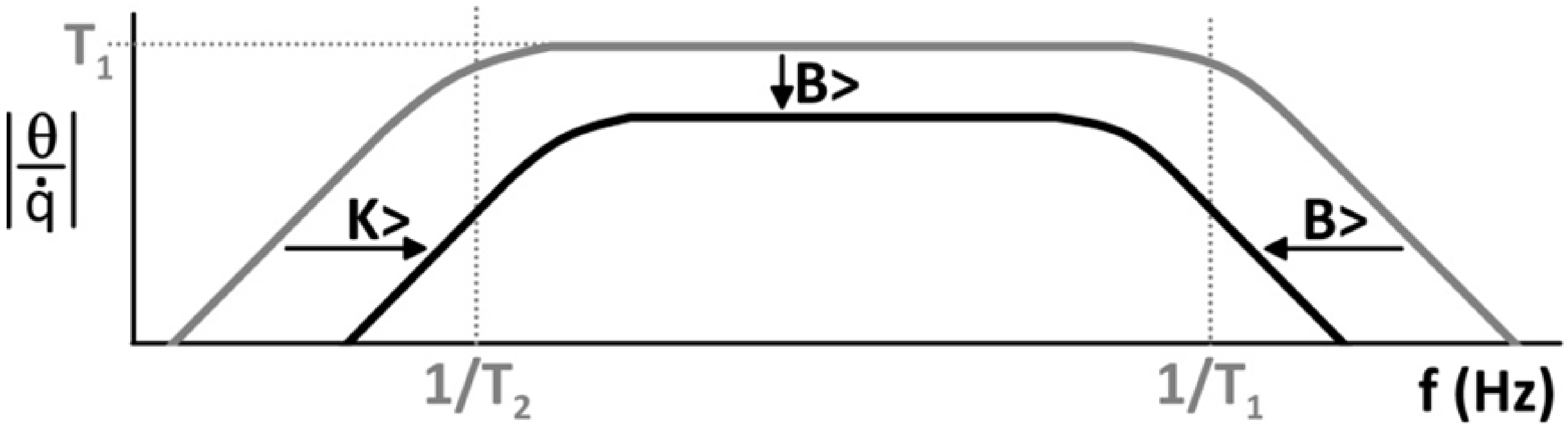
canal senses acceleration, cupula deflection indicates head velocity

frequency dependence canals: phase (≈ time constant)



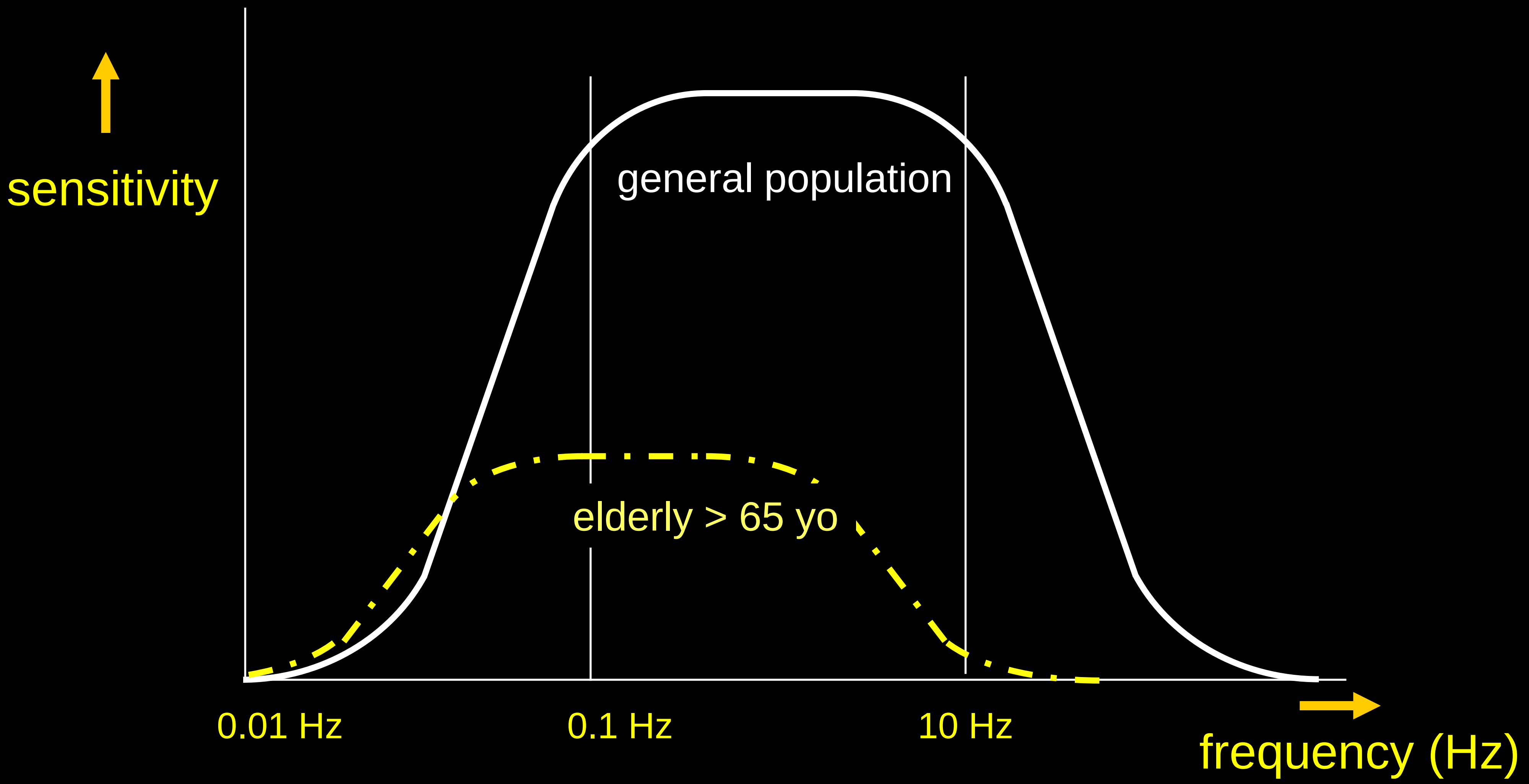


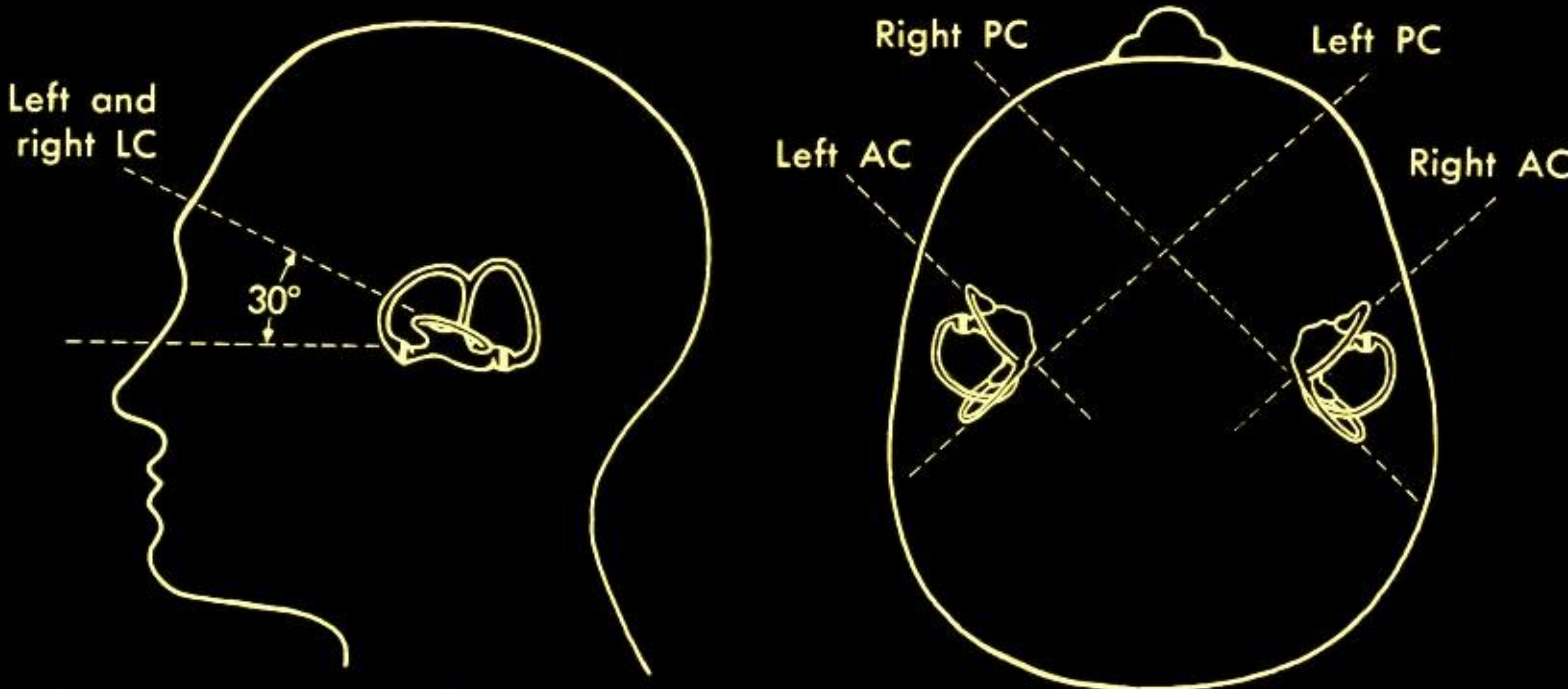
impact viscosity B and elasticity K on canal function



- mechanical changes
  - viscosity B
  - elasticity K
  - specific mass (e.g. alcohol intake, canaloliths)

ageing (>60) frequency dependence canals presbyo-vertigo





A B

quantification of labyrinth function

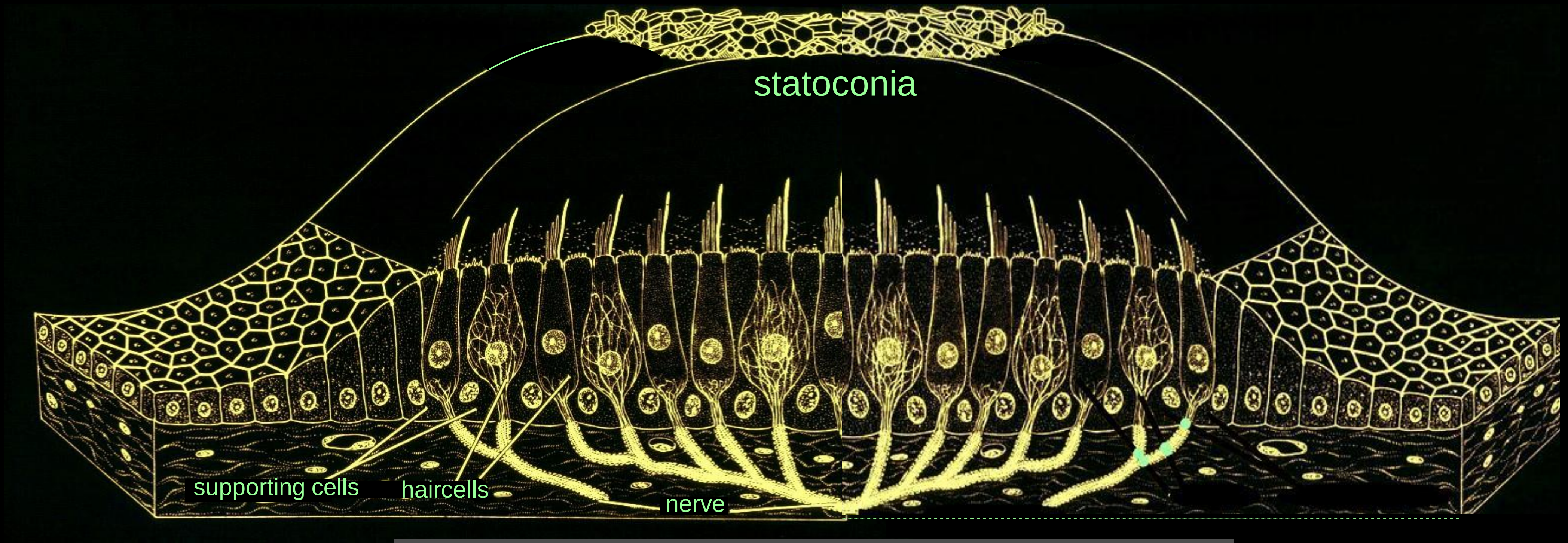
two labyrinths

- horizontal canal
- anterior canal
- posterior canal
- utriculus
- sacculus

labyrinth

• rotations: canal system

• translations + tilt: statolith systems



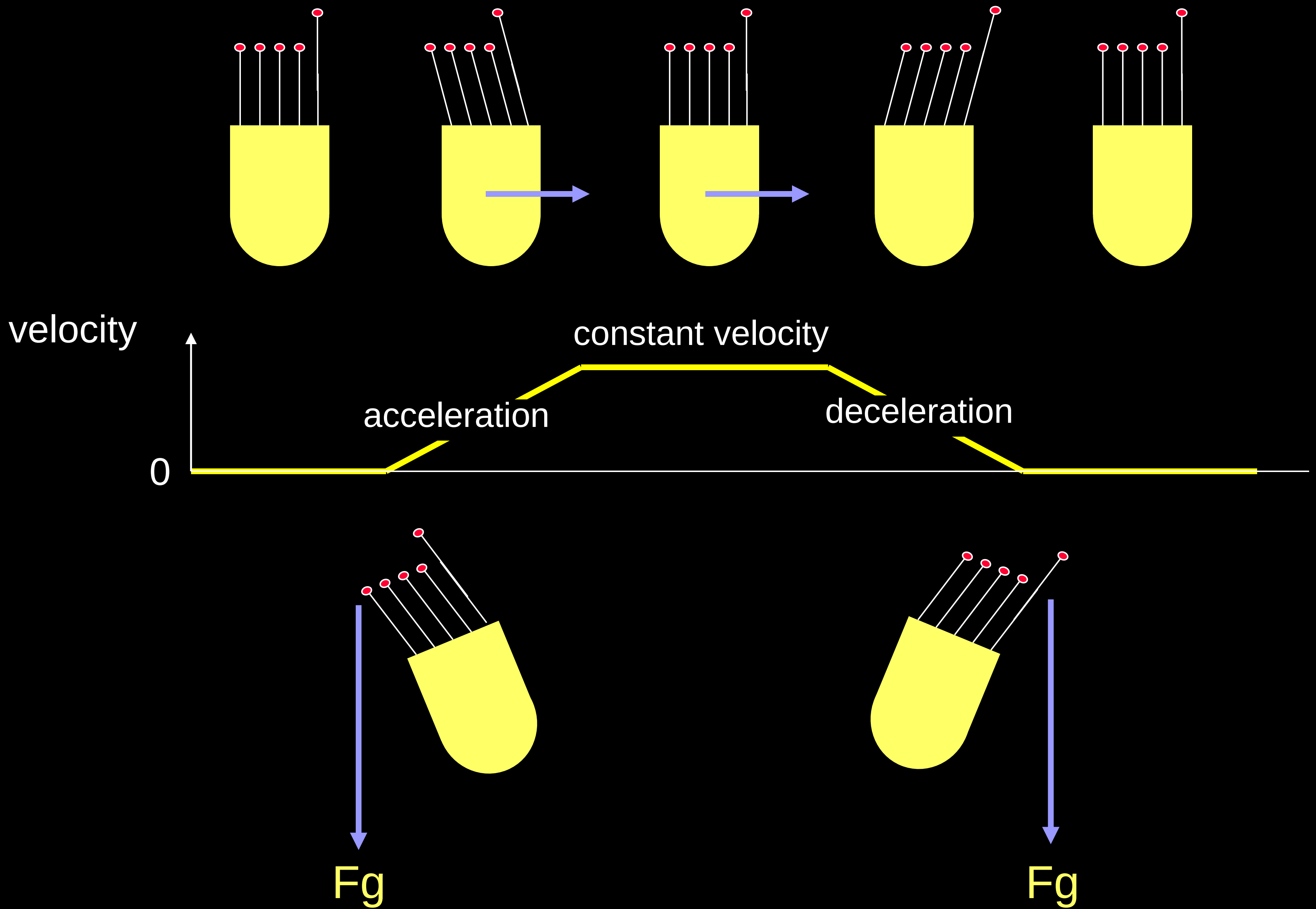
utriculus + sacculus

accelerometers

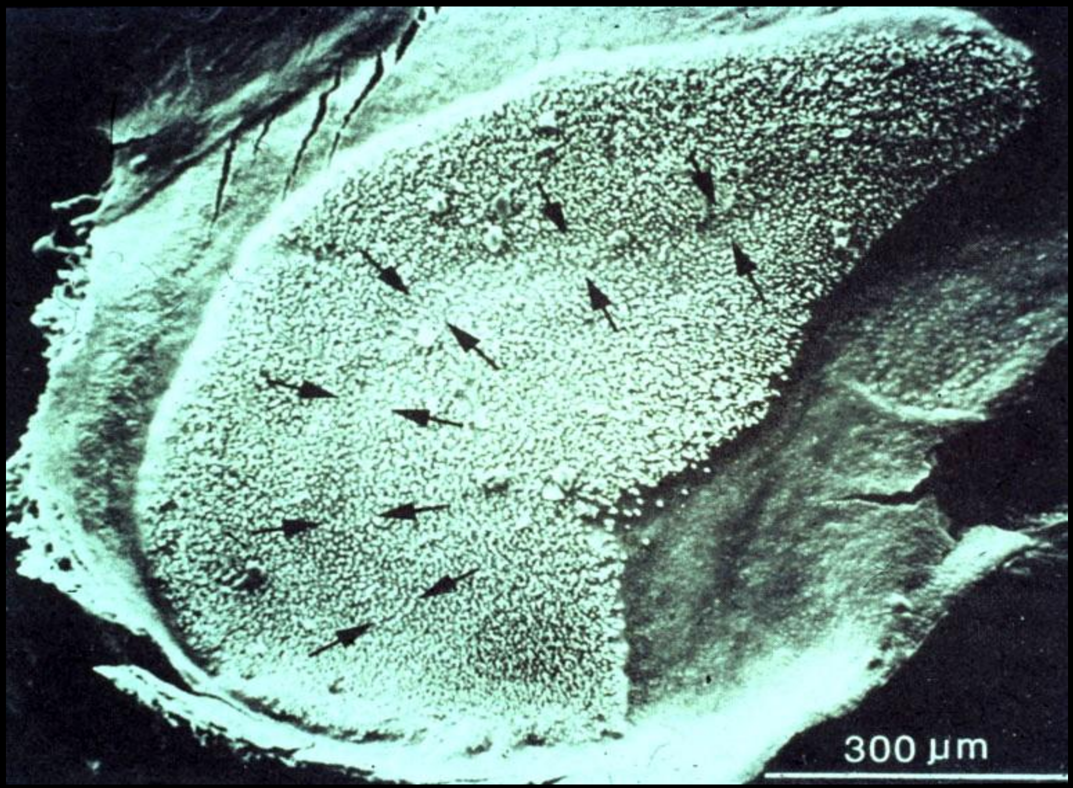
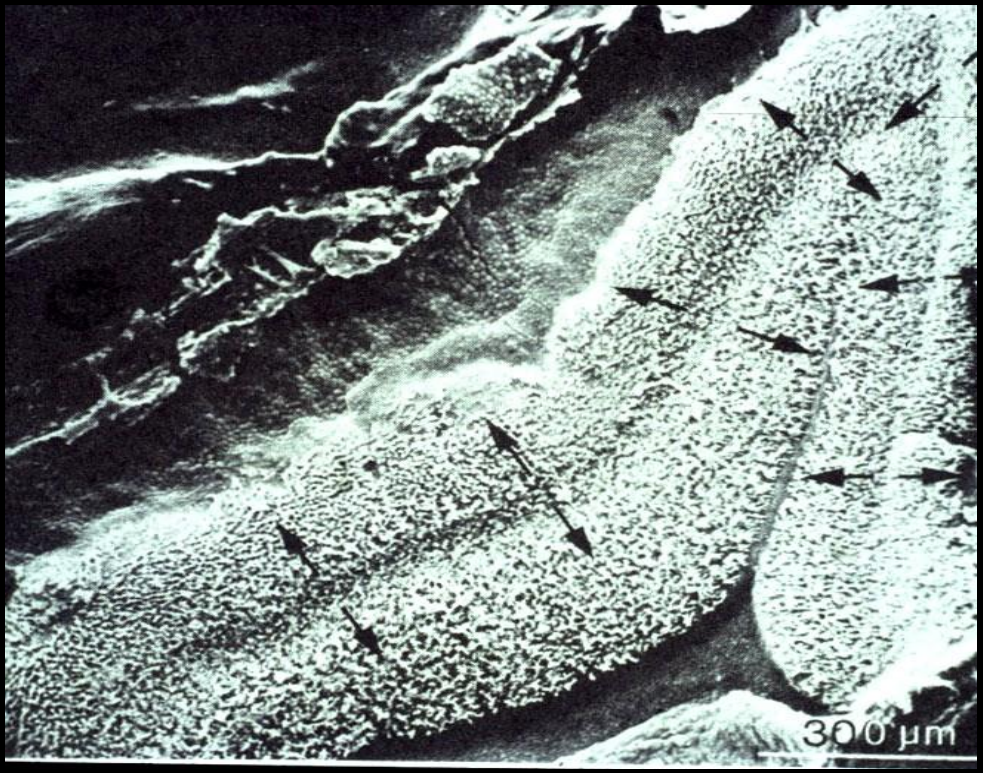
• function based on inertia of statoconia mass

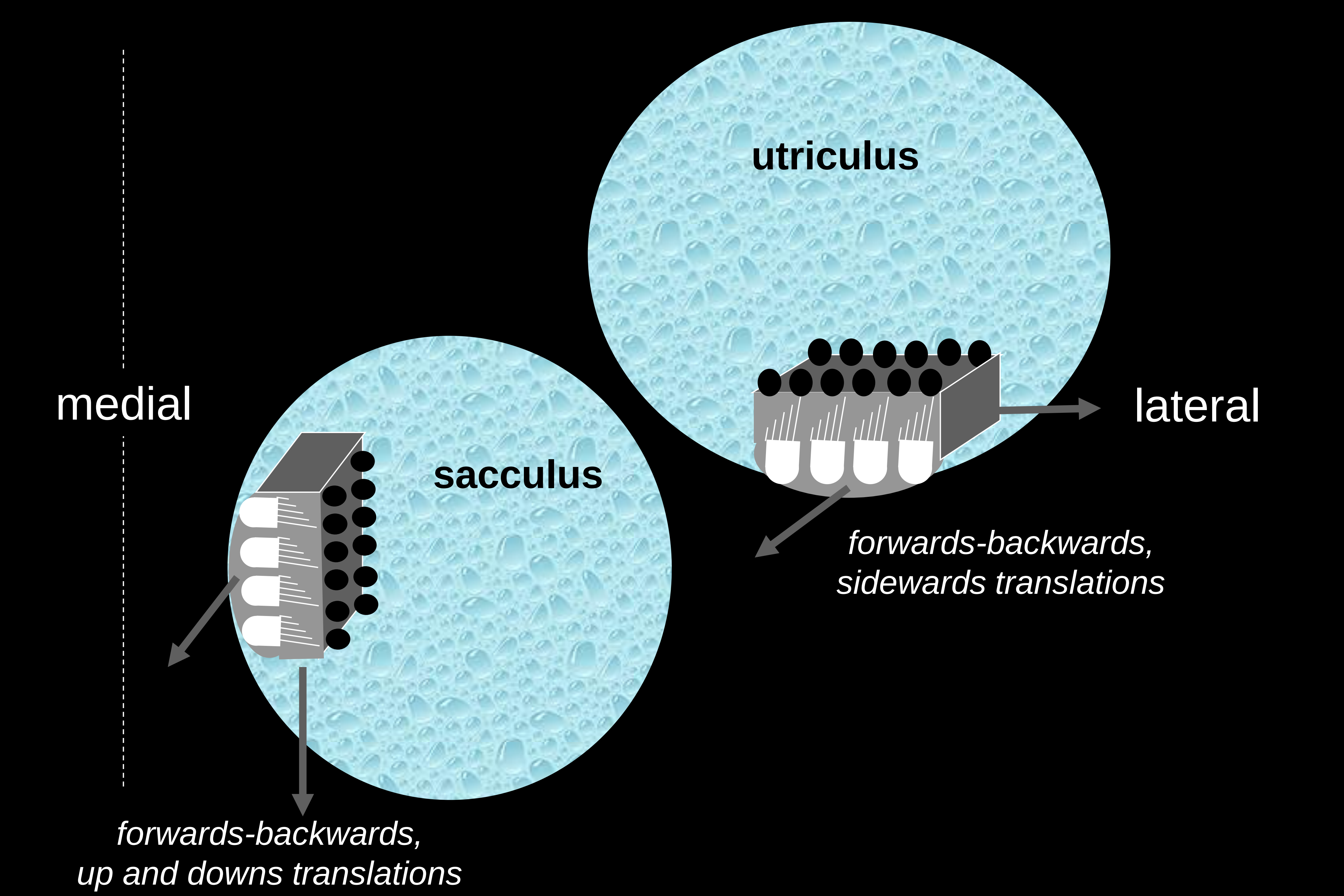
• multi-directional symmetrical sensitivity

• frequency dependence

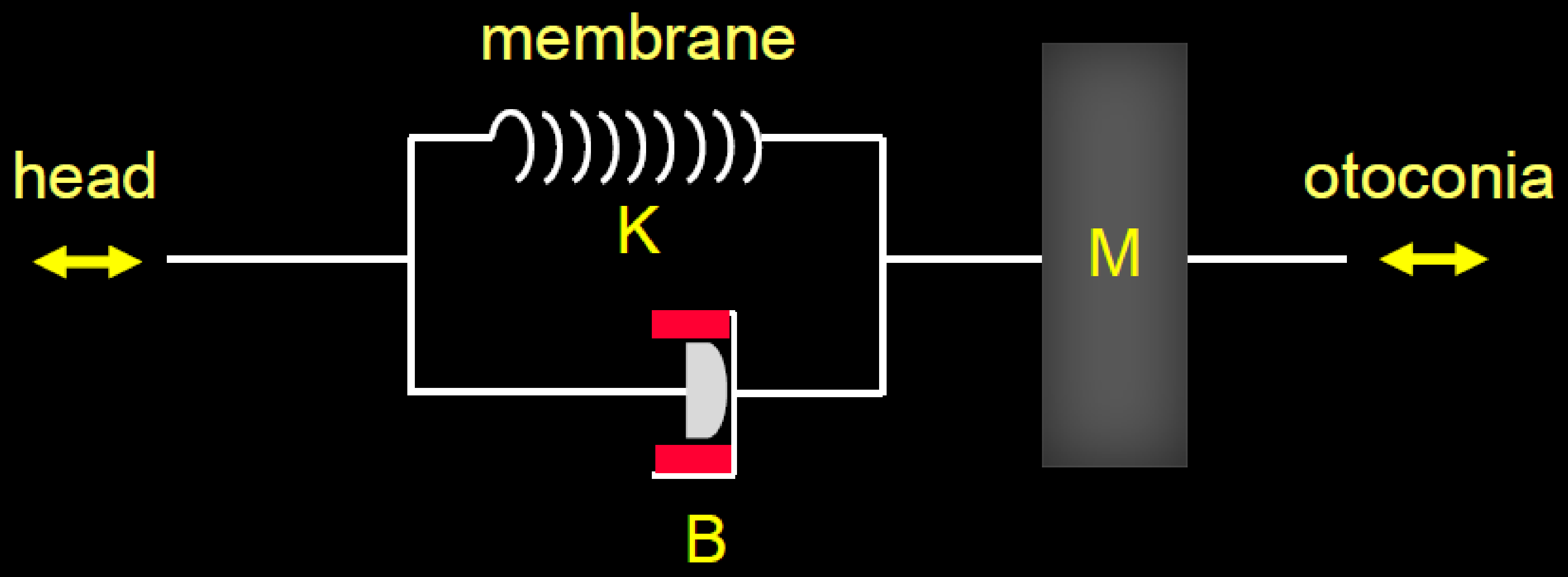


no discrimination between translation and tilt possible


300 µm

300 µm


utriculus
medial
lateral
sacculus
forwards-backwards,
sidewards translations
forwards-backwards,
up and downs translations

theoretical model otolith membrane: again 2nd order system



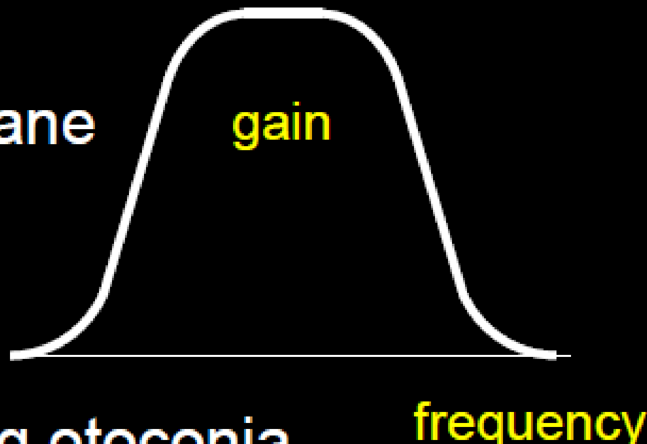
M ~ otoconia mass
B ~ friction (viscosity)
K ~ elasticity otoconia-membrane

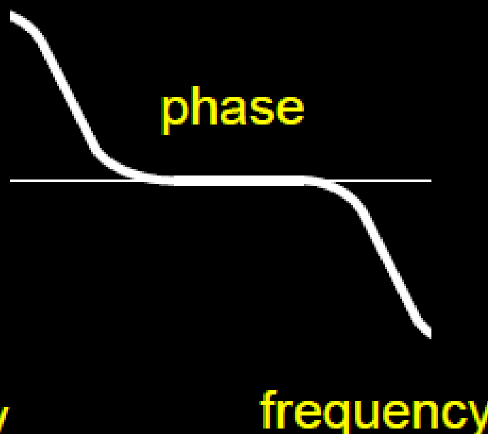
theoretical model otolith membrane: 2nd order system

leads to the following differential equation

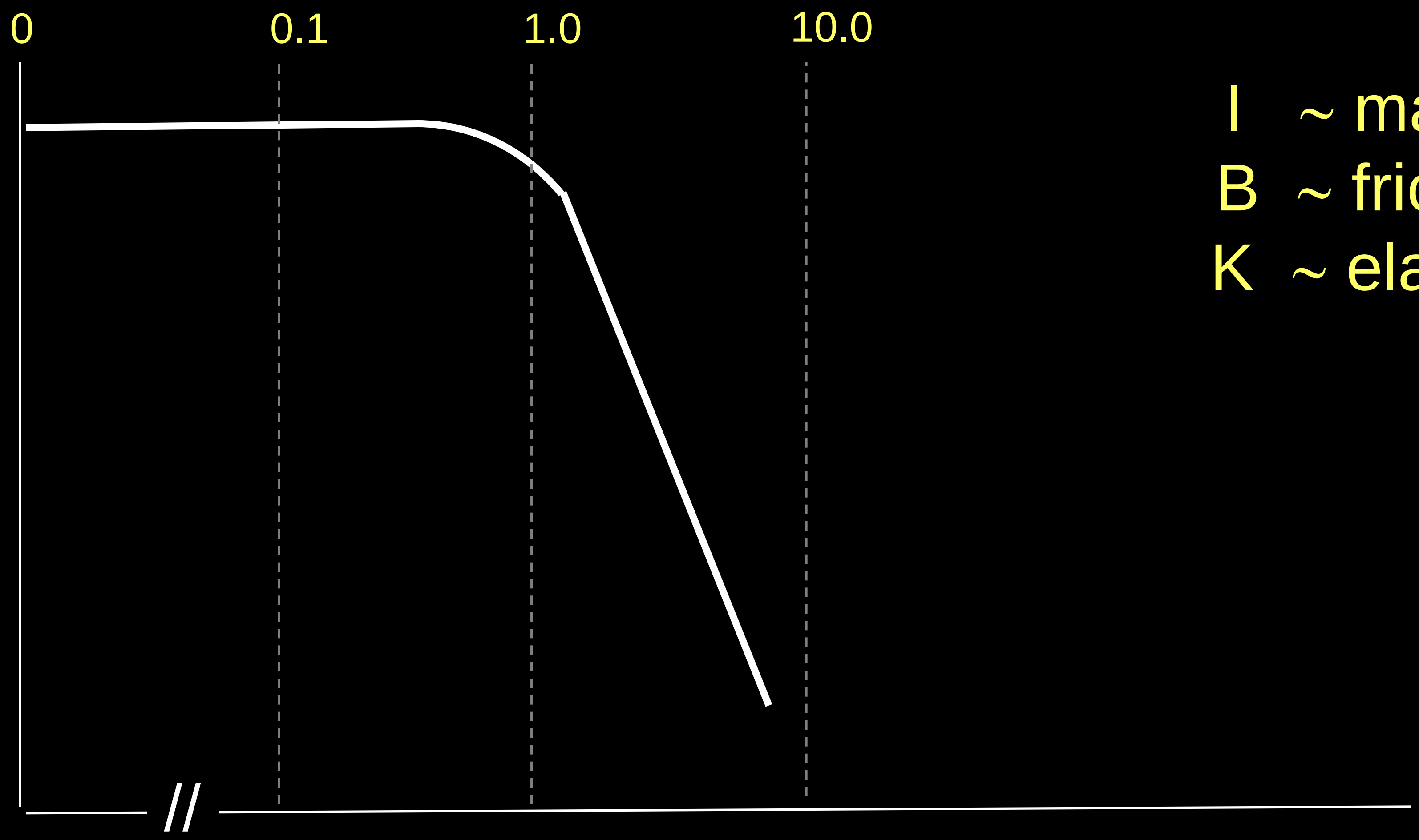
$$\left(1-\frac{\rho_e}{\rho_o}\right)\ddot{x} = \Delta\ddot{x} + \frac{B}{M}\Delta\dot{x} + \frac{K}{M}\Delta x$$

$\ddot{X}$ acceleration head
Δx relative displacement membrane
M ~ otoconia mass
B ~ friction (viscosity)
K ~ elasticity otolith membrane
ρ_e and ρ_o density endolymphe cq otoconia





gain = membrane shift / head acceleration



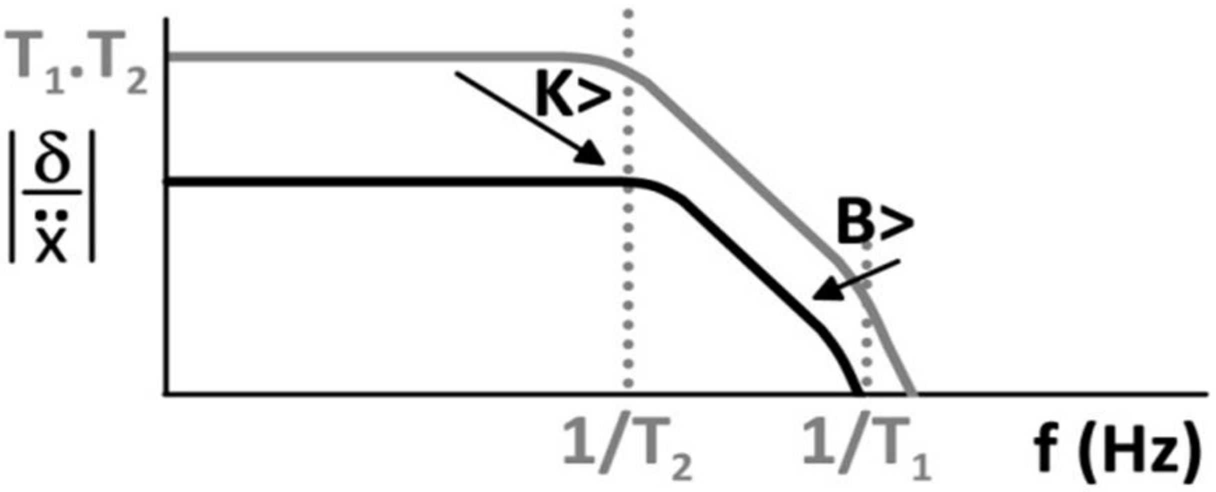
I ~ mass
B ~ friction (visc)
K ~ elasticity

frequency (Hz)

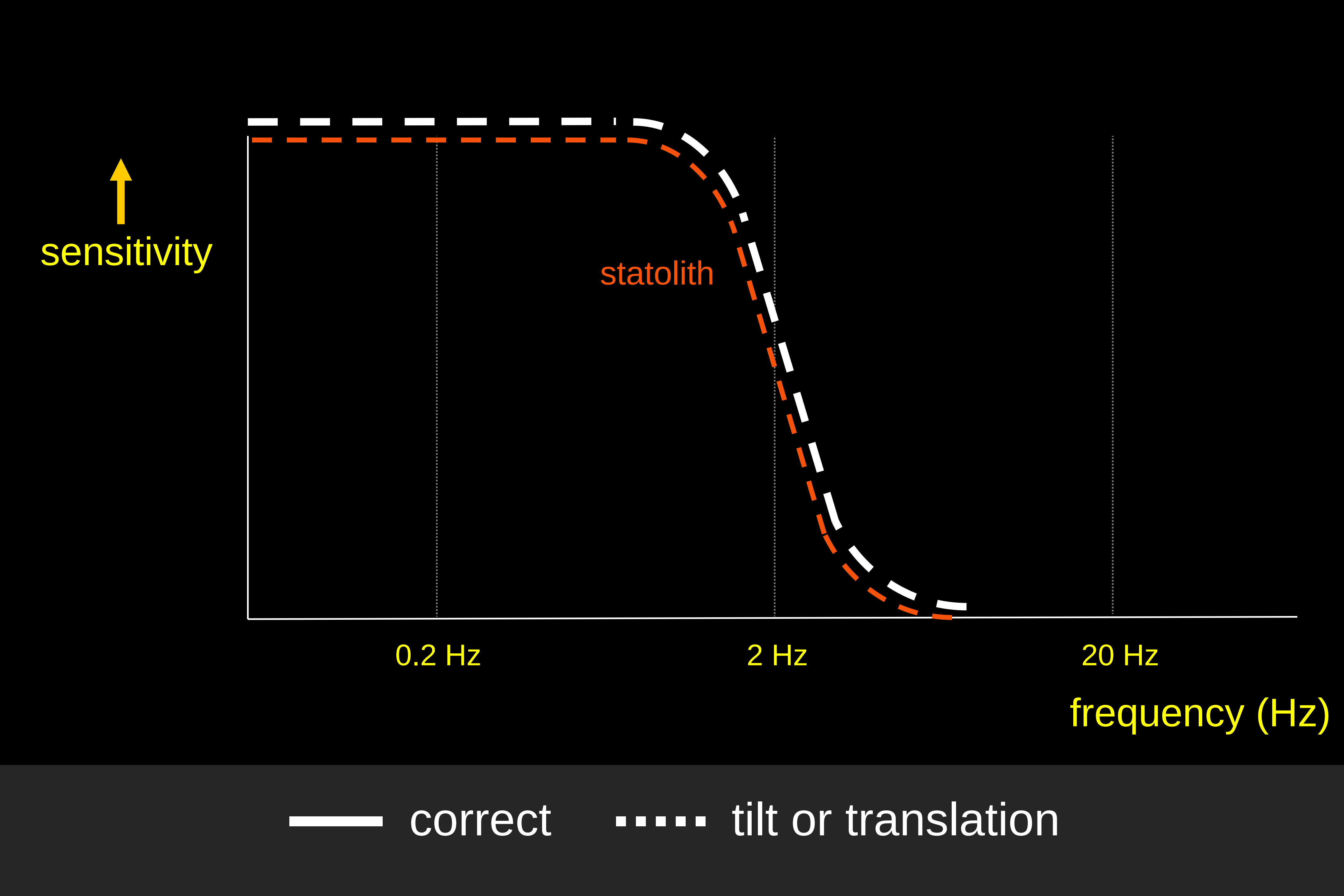
$1 / T_{low} = \dfrac{K}{B}$ $\quad 1 / T_{high} = \dfrac{B}{I}$

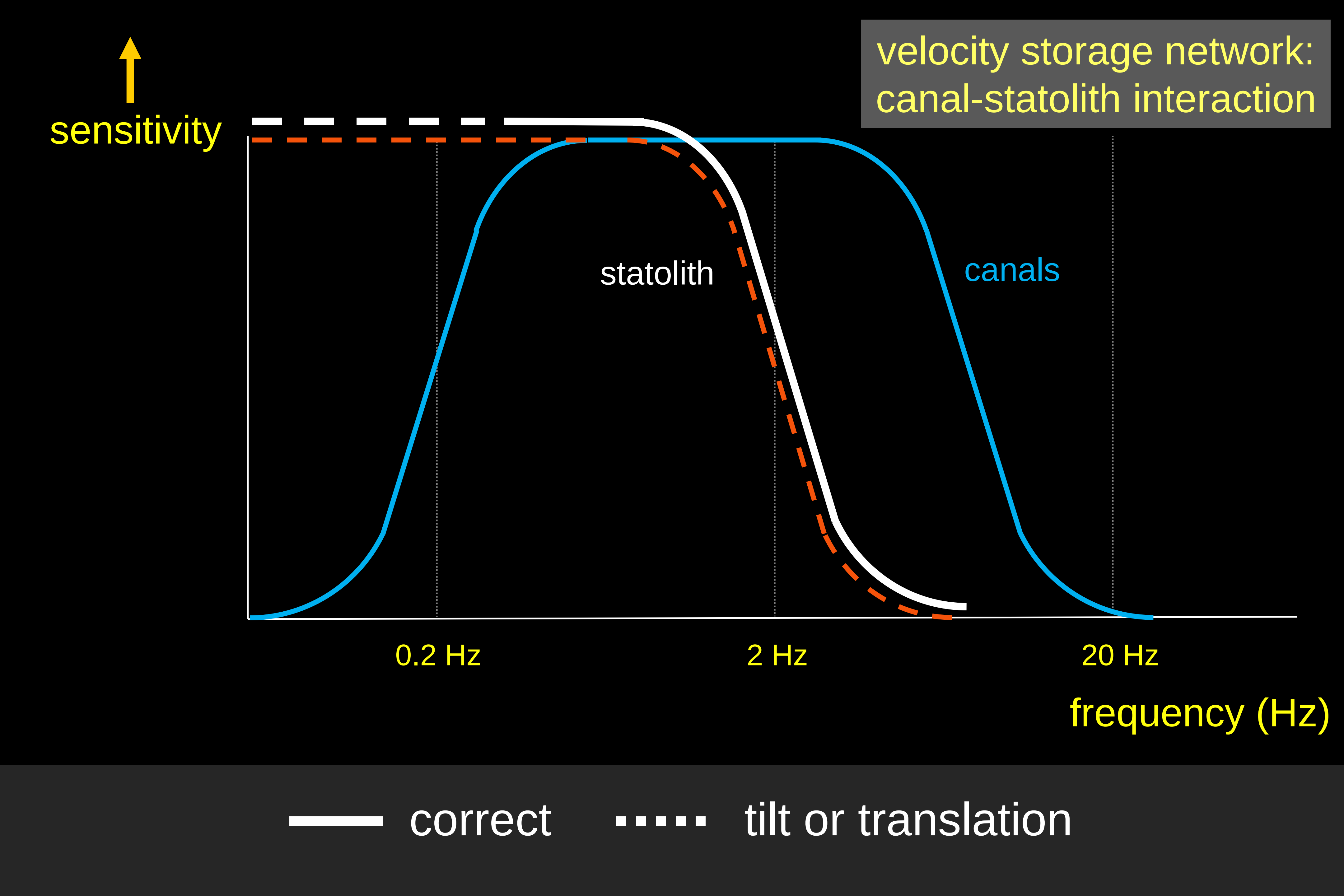
optimal sensitivity for the gravity vector

impact viscosity B and elasticity K on statolith function

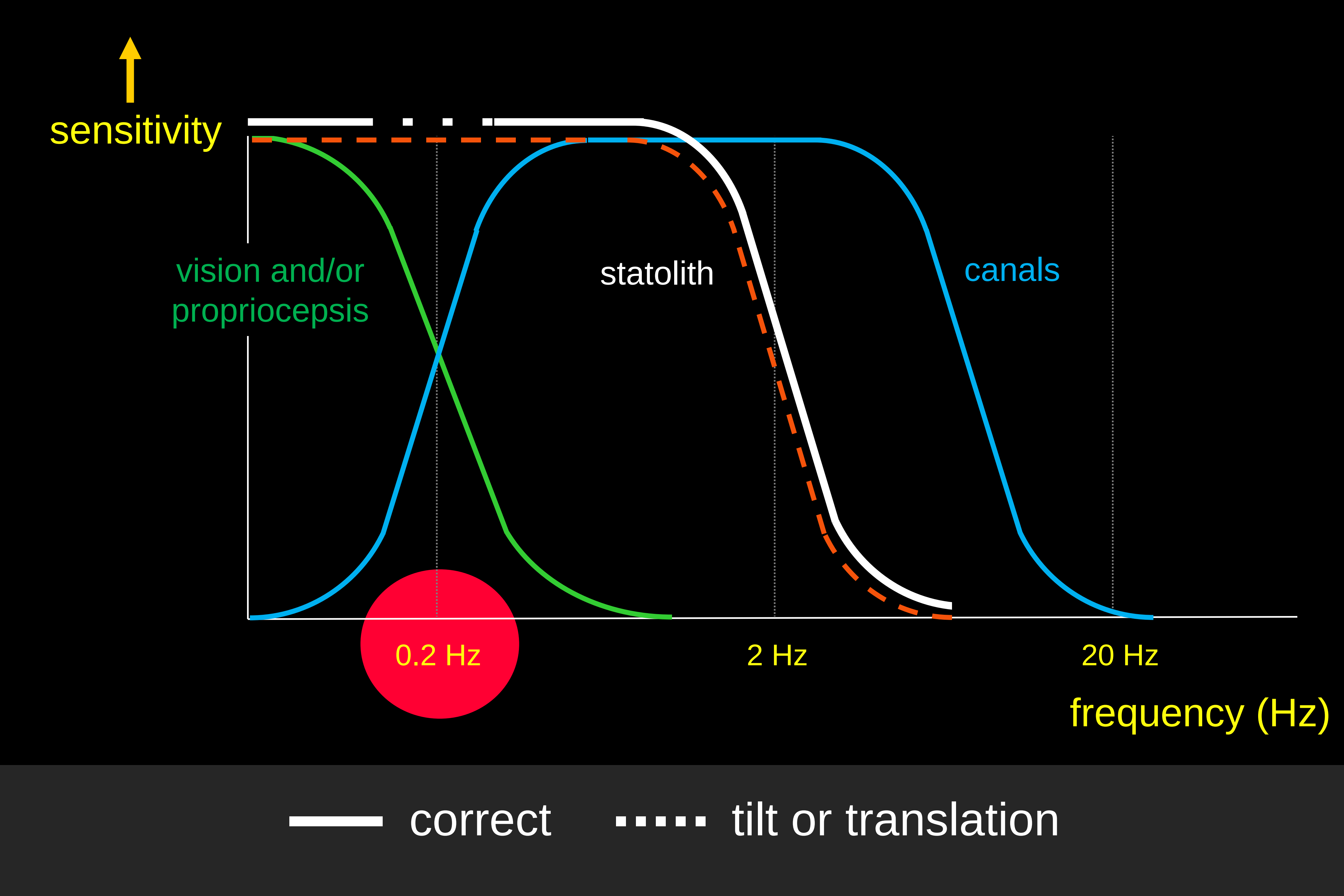


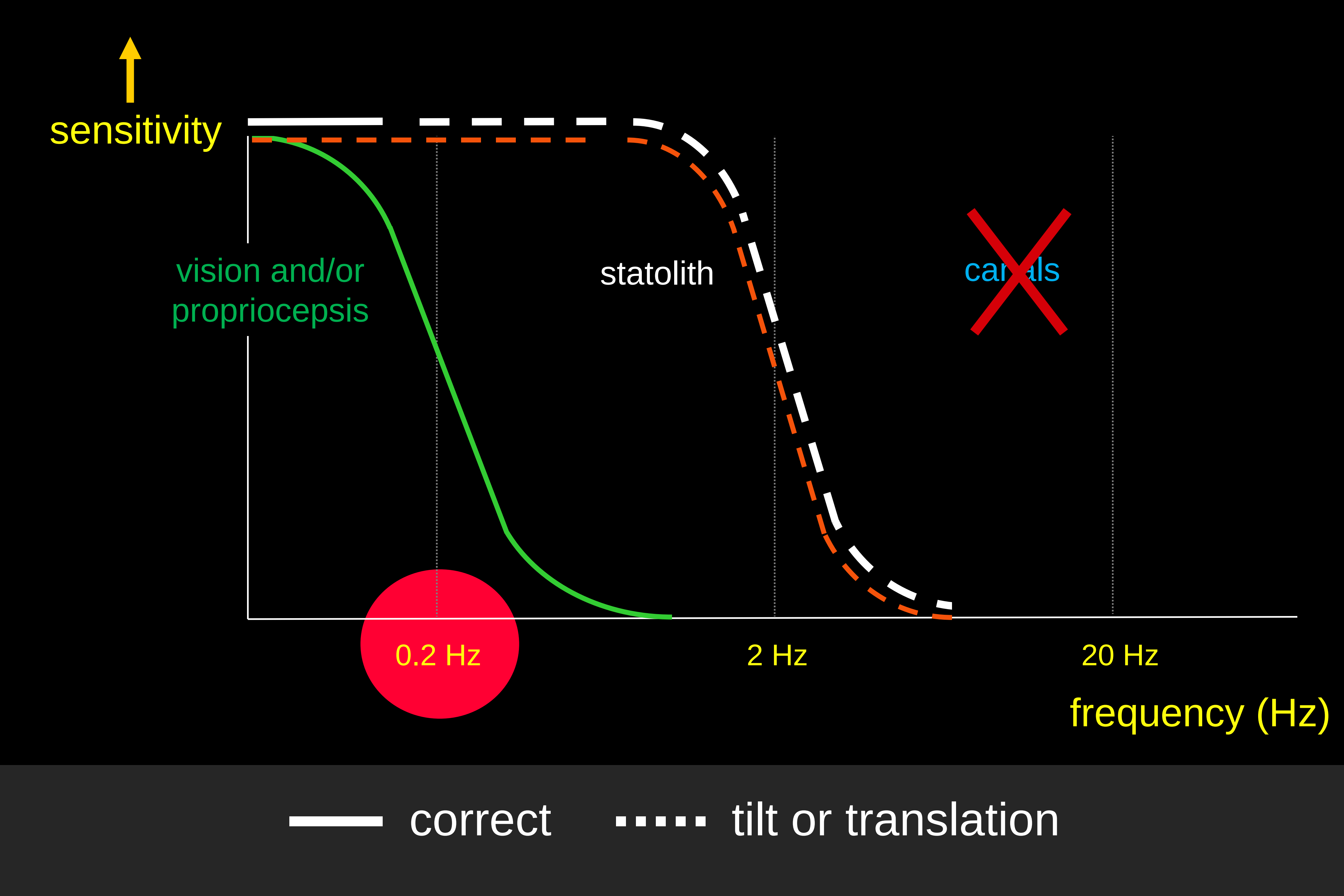
- mechanical changes
  viscosity B
  elasticity K
  specific mass otoconia: gain ↓


sensitivity
statolith
0.2 Hz
2 Hz
20 Hz
frequency (Hz)
correct
tilt or translation



velocity storage network:
canal-statolith interaction
sensitivity
statolith
canals
0.2 Hz
2 Hz
20 Hz
frequency (Hz)
correct
tilt or translation


sensitivity
vision and/or
propriocepsis
statolith
canals
0.2 Hz
2 Hz
20 Hz
frequency (Hz)
correct
tilt or translation



sensitivity
vision and/or
proprioceptis
statolith
canals
0.2 Hz
2 Hz
20 Hz
frequency (Hz)
correct
tilt or translation

some facts and findings that need to be explained

- divers under water can’t orient themselves without vision !

  submersion in water:

  principle of inertia of mass in labyrinth remains

  → normal detection of accelerations should be possible

- no detection of orientation when covered by an avalanche

so: the brain needs multi-sensory input or pre-knowledge

otherwise statolith input is neglected:

……falling asleep

which complaints are related to vestibular deficits ?

which complaints are related to natural limitations ?

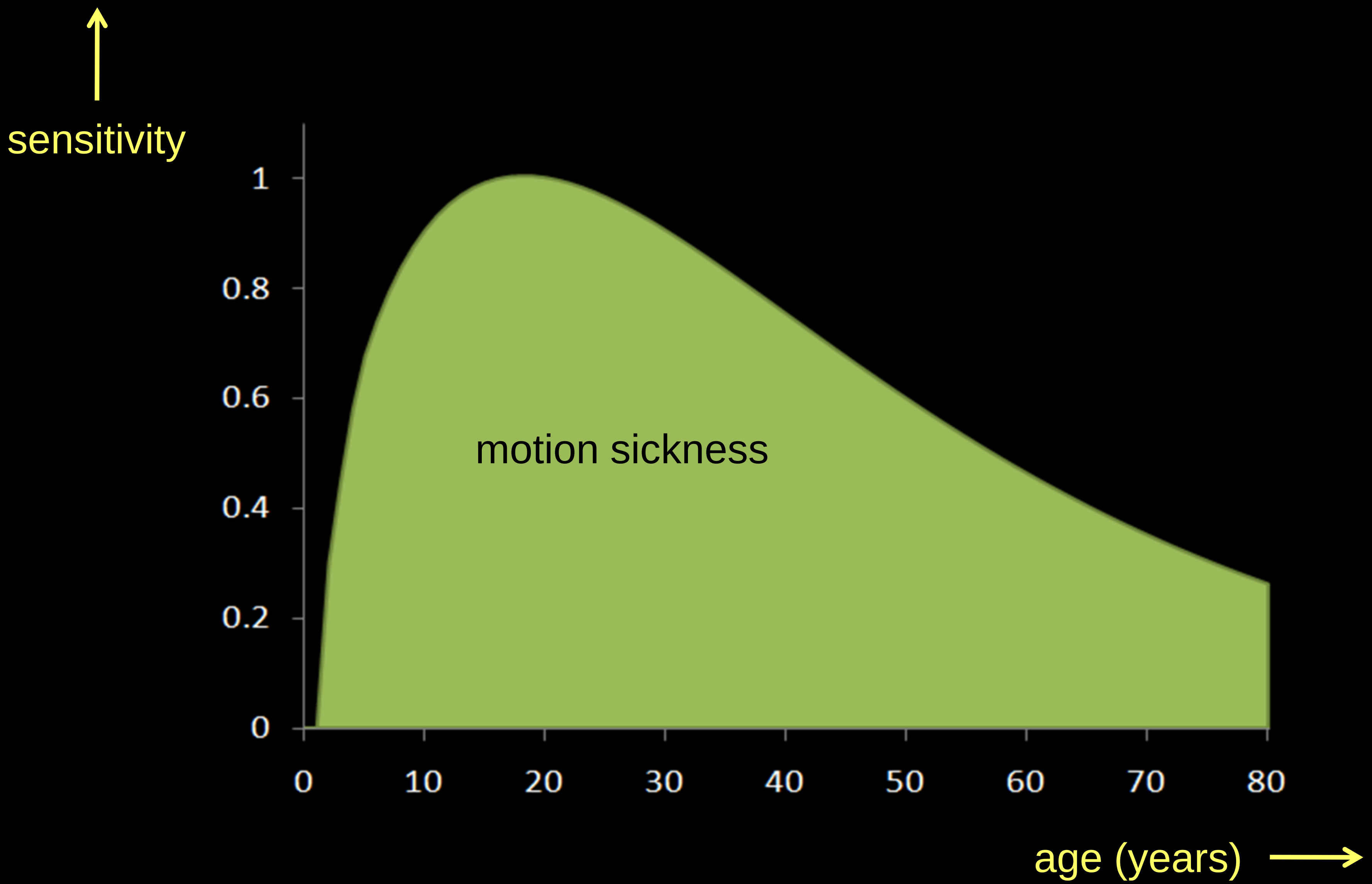
canals:	orientation in space: constant rotation or stand still ?
statoliths:	orientation in space: constant translation or stand still ?
	orientation relative to gravity: tilt or translation ?

when correct interpretation fails (gravity / selfmotion)

motion sickness

- almost all subjects are susceptible with correct stimulus
  unless a low neuro-vegetative sensitivity
  training / adaptation helps

- a (partly) working labyrinth is prerequisite for Motion Sickness:


sensitivity
1
0.8
0.6
0.4
0.2
0
motion sickness
0
10
20
30
40
50
60
70
80
age (years)

superior nerve

horizontal scc

anterior scc

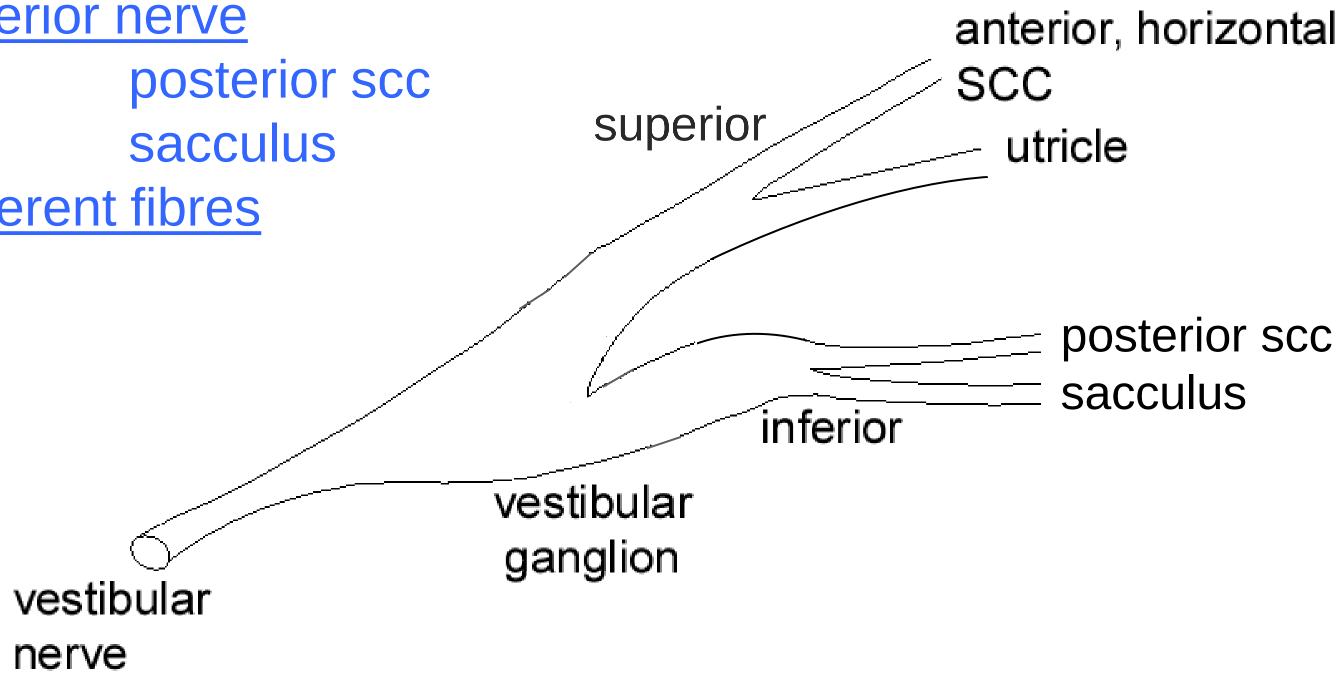
utriculus

inferior nerve

posterior scc

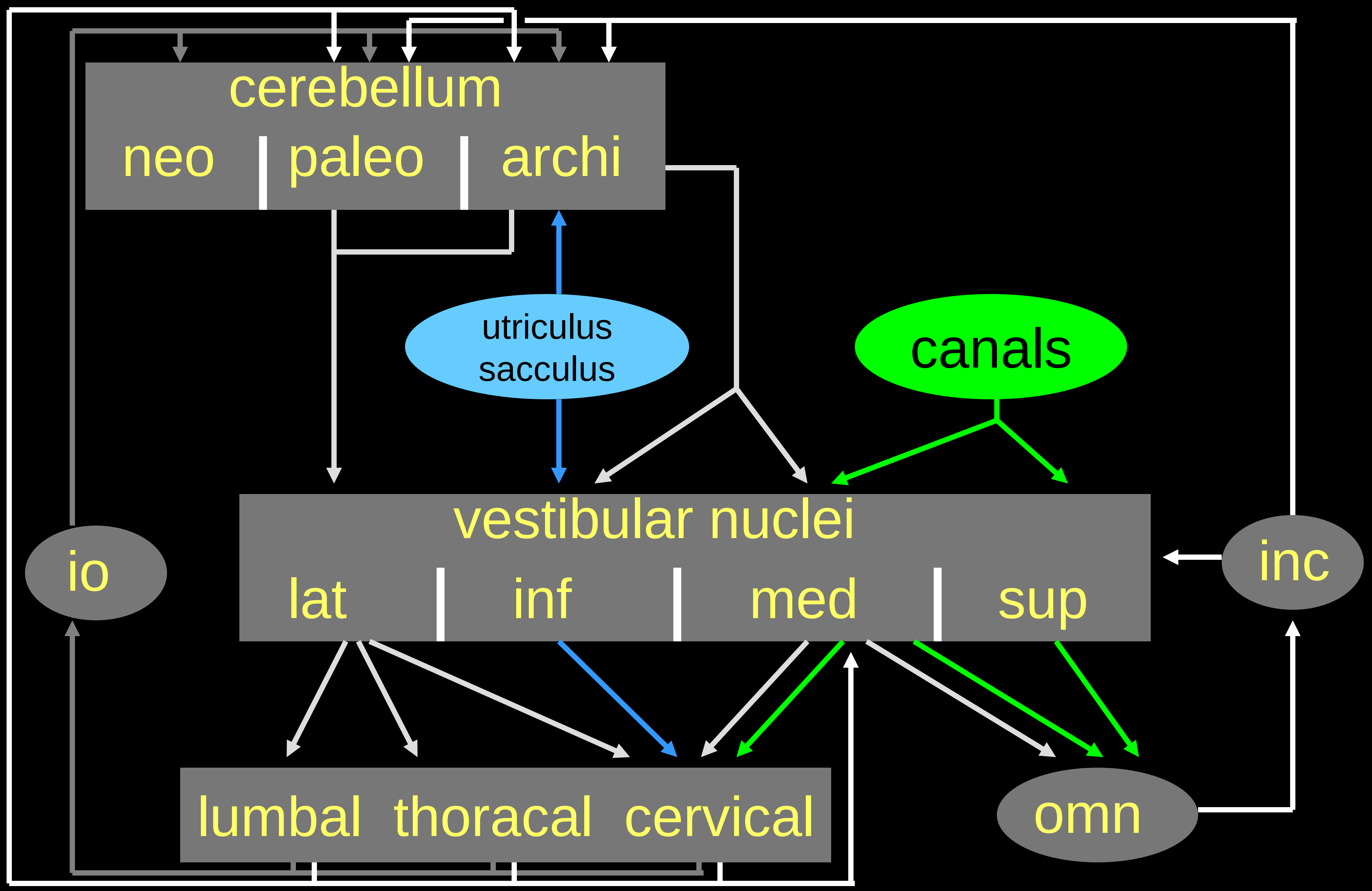
sacculus

efferent fibres



many hair cells receive efferent input

the brain controls the periphery


cerebellum
neo
paleo
archi
utriculus
sacculus
canals
io
vestibular nuclei
lat
inf
med
sup
inc
lumbal
thoracal
cervical
omn

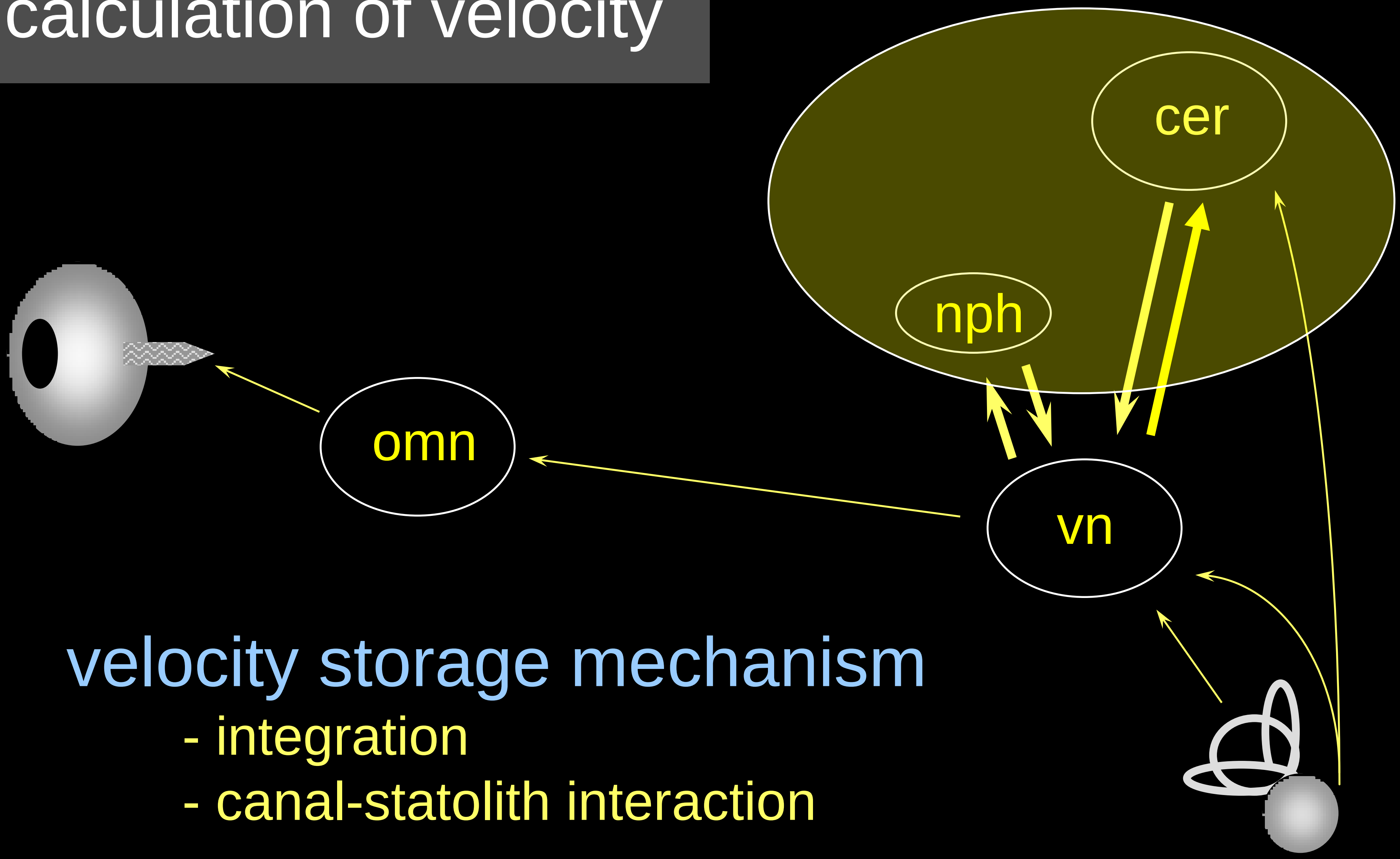
memories and integration in the brain of signals from the labyrinth (accelerometer)

aim:

- image stabilisation after head motion
- increase of sensitivity
- calculation of head velocity

- increase of sensitivity
- calculation of velocity

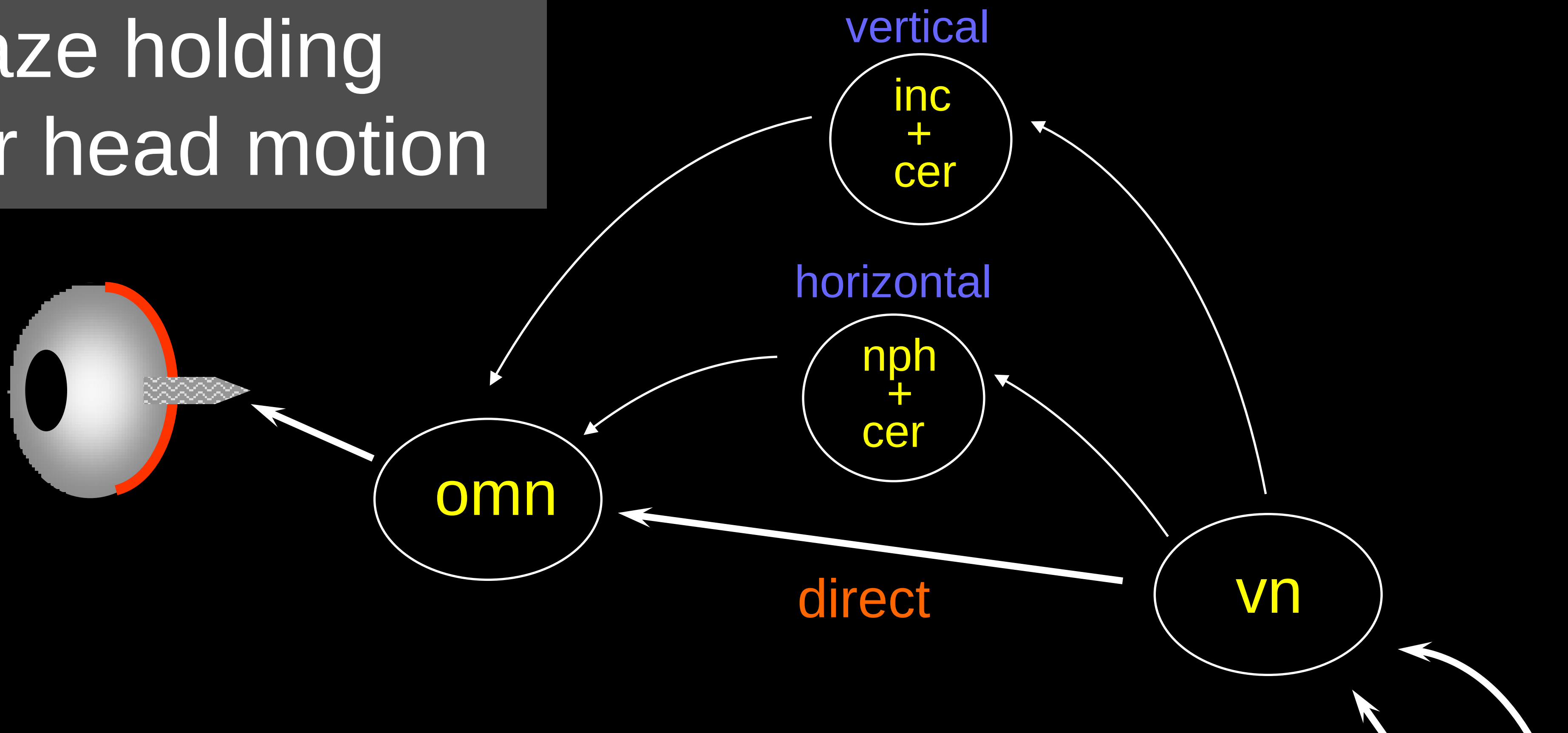
duration 20 s ⇒ 60 s

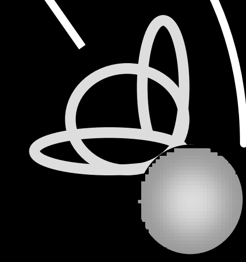


velocity storage mechanism

- integration
- canal-statolith interaction

gaze holding after head motion



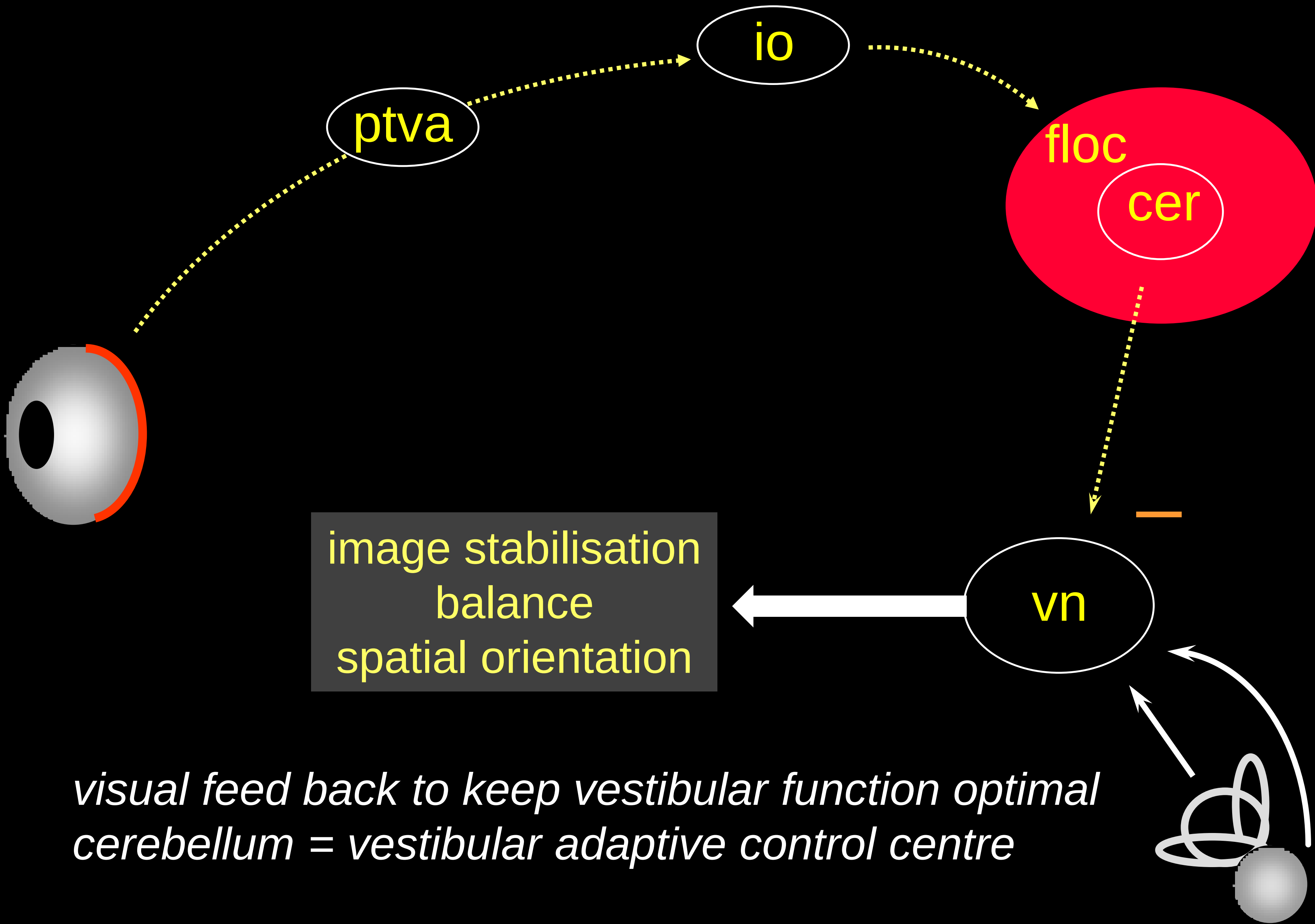


VOR

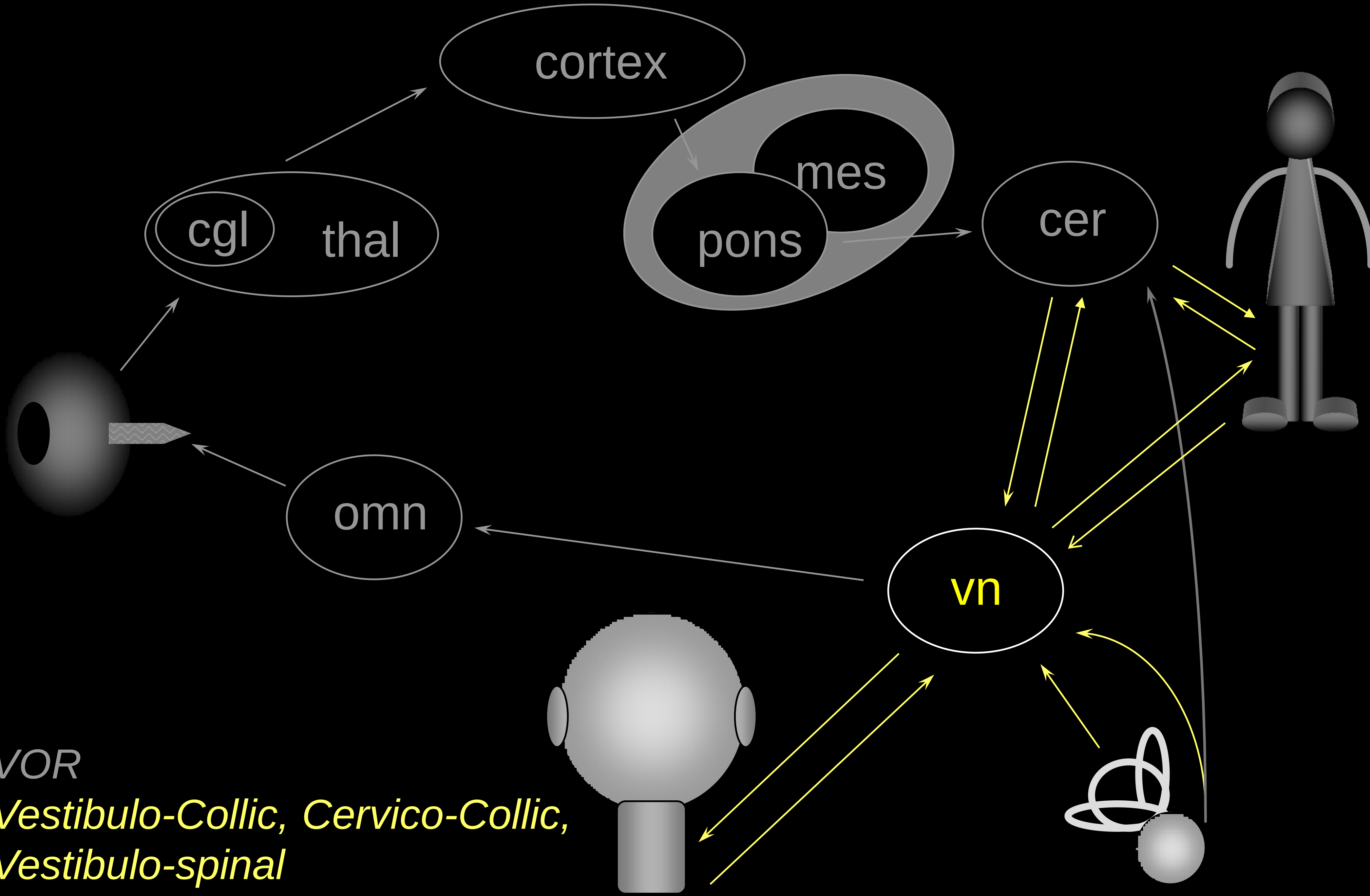
direct: to compensate during head motion

indirect: gaze holding: to keep the eye for 100 ms on target after head motion before the slow visual fixation can take over

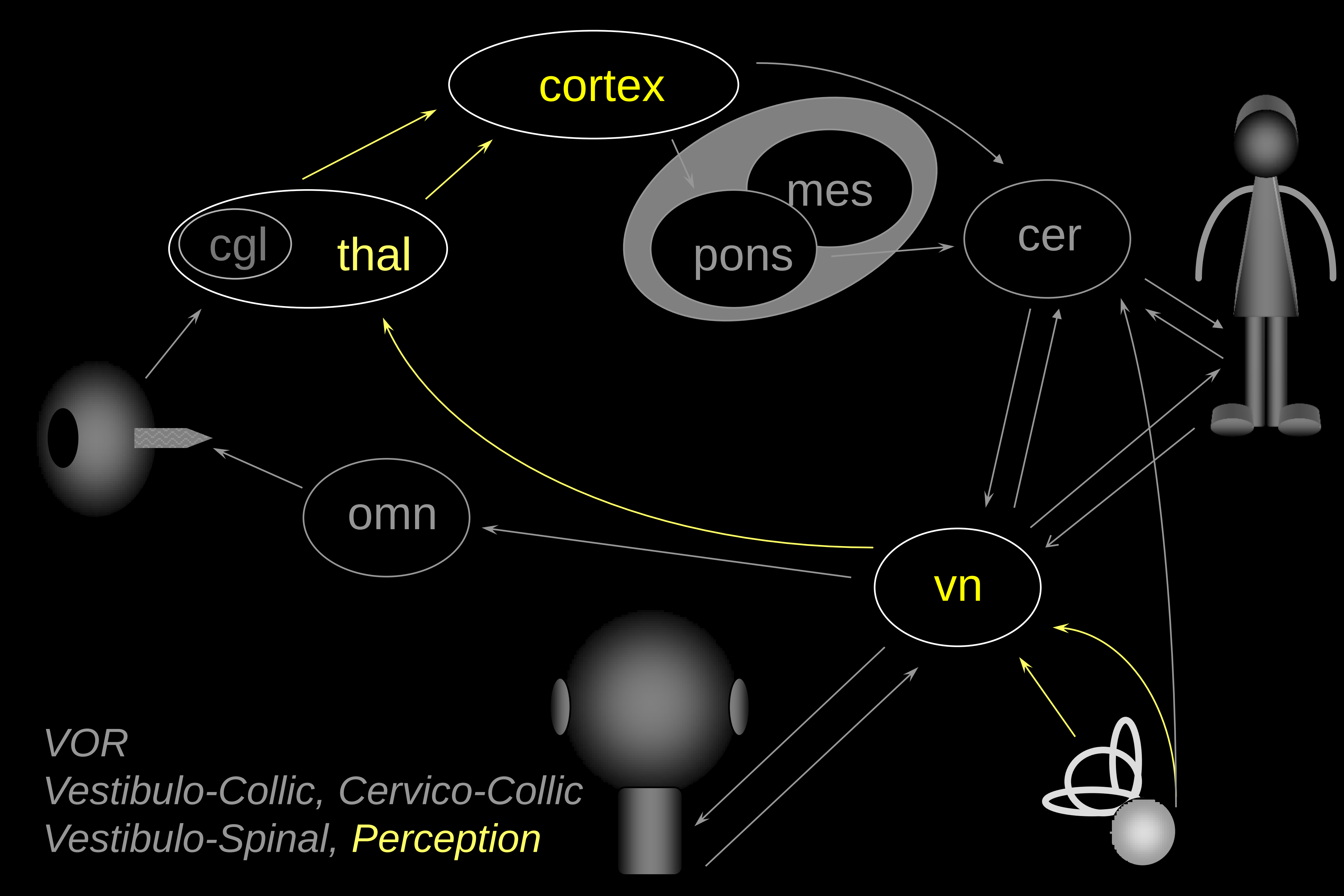
pathology: gaze evoked nystagmus

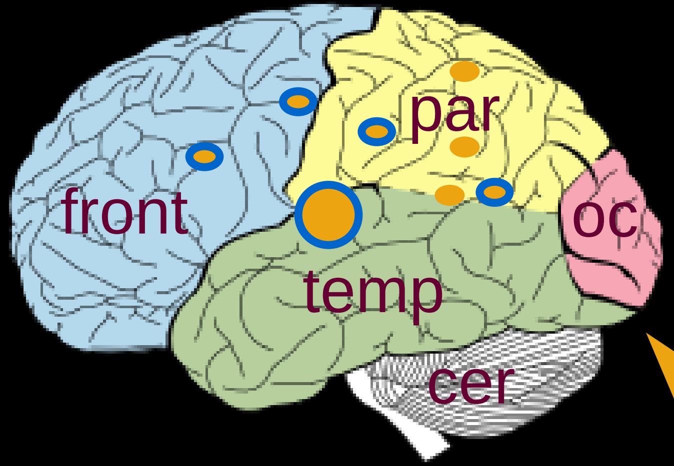


visual feed back to keep vestibular function optimal
cerebellum = vestibular adaptive control centre



VOR
Vestibulo-Collic, Cervico-Collic,
Vestibulo-spinal


cortex
mes
pons
cgl
thal
cer
omn
vn
VOR
Vestibulo-Collic, Cervico-Collic
Vestibulo-Spinal, Perception



- dominance right vestibular hemisphere
  respective side of labyrinth stimulation

- PIVC activation: parallel deactivation of
  occipital and parietal visual areas and vv

- efferent projections

perception: cortical network

temporo-insular and temporo-parietal cortex

parieto-insular vestibular cortex (PIVC)

retro-insular cortex

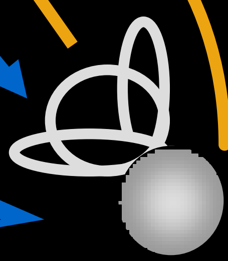
superior temporal gyrus (STG)

inferior parietal lobule (IPL)

precuneus

anterior cingulum

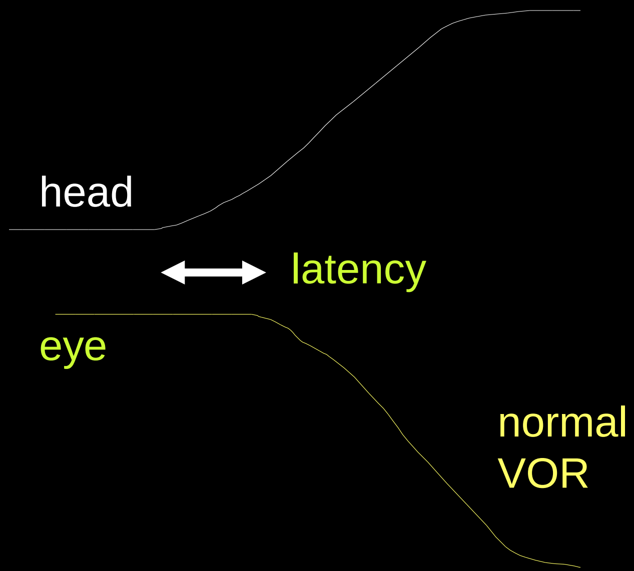
hippocampus



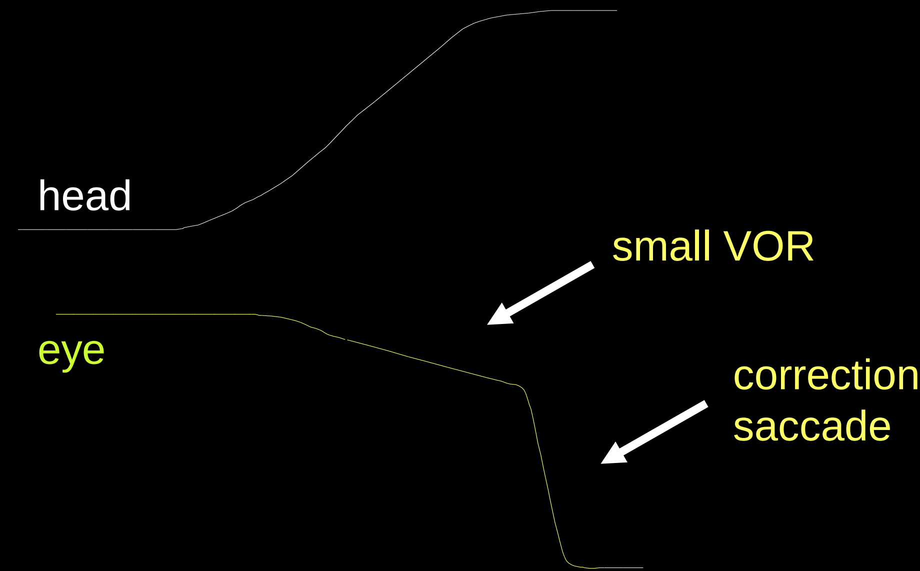
thank you for your kind attention

EyeSeeCam® (München)

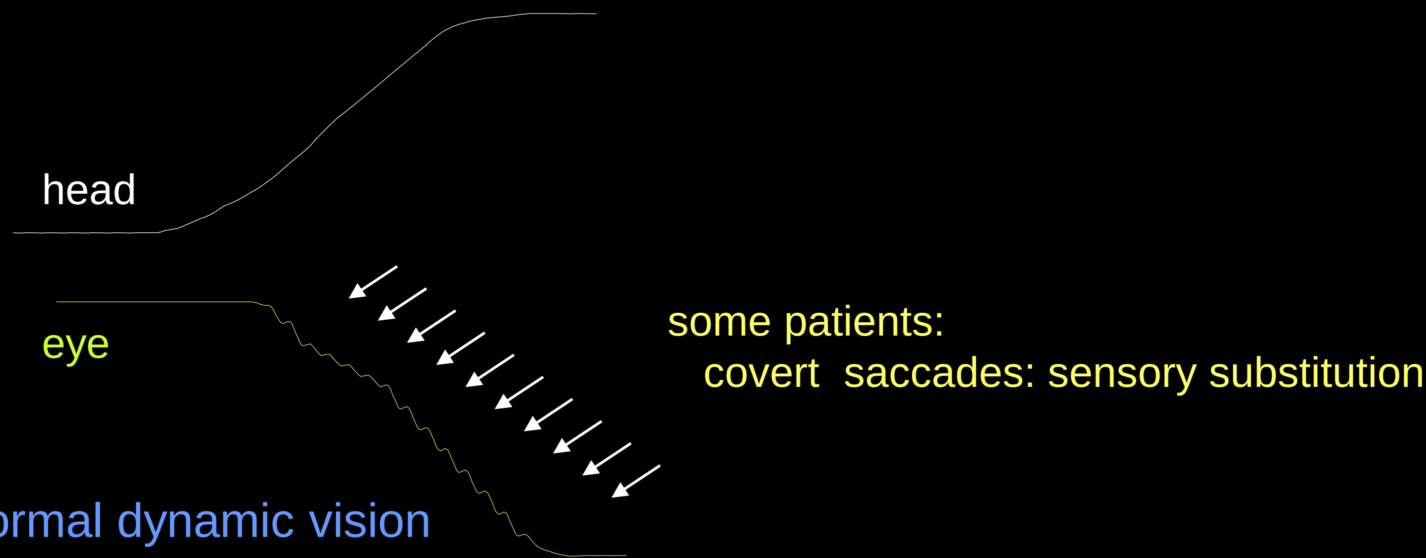
ICS Impulse® (Sydney)



normal dynamic vision
normal head impulse test



poor dynamic vision
abnormal head impulse test



normal dynamic vision
seemingly normal head impulse test

unilateral or bilateral peripheral vestibular loss

head impulse test

- often 1-2 big correction saccades
- some patients compensate with many covert saccades

normal test by observation: does not exclude function loss