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 unilateral** loss or fluctuating function (neuritis, Ménière…)

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

**Acute bilateral** 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?
which complaints are related to vestibular deficits?

which complaints are related to natural limitations?
image stabilisation

balance control

labyrinths

CNS
interpretation
learning
adaptation
compensation

gravitoreceptors
blood pressure sensors in large blood vessels

vision

hearing

circadian rhythm
vestibular projections hypothalamus supra-chiasmatic nucleus

vision

labyrinths

CNS
interpretation
learning
adaptation
compensation

autonomic processes
fast blood pressure regulation heart beat frequency nausea / vomiting

Vestibular effects on cerebral blood flow Serrador et al, BMC Neuroscience 2009

blood pressure sensors in large blood vessels

CNS
interpretation
learning
adaptation
compensation

spatial orientation

Vestibular effects on cerebral blood flow Serrador et al, BMC Neuroscience 2009

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
image stabilisation

labyrinths

gravitoreceptors
blood pressure sensors in large blood vessels

autonomic processes
blood pressure regulation
heart beat frequency
respiration rate
nausea / vomiting

CNS
interpretation
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circadian rhythm
vestibular projections
hypothalamus
supra-chiasmatic nucleus

somatosensory
e.g. foot sole pressure

平衡控制

空间定向

学习

补偿

脑干

视网膜

眼动调节

心率

呼吸频率

恶心 / 呕吐

视觉

听觉

生物节律

前庭投射

下丘脑

上丘脑的核团

自主性过程

血压调节

脚底压力
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*
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 feedback

fast tandem walk
   using visual anticipation
complaints related to vestibular dysfunction

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transient: vertigo, nausea, falling / imbalance

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spatial orientation

balance control
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

normal values (maximum VA decrease)

- 0.2
- 0.2
- 0.3

BV Patients
Healthy Subjects
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
- reduced automatisation of balance
- reduced dynamic visual acuity
- reduced perception of self motion
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which complaints are related to vestibular deficits?

which complaints are related to natural 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)
Ewald’s 2\textsuperscript{nd} Law: asymmetry

- Myosine filaments
- Ion channels
- Action potentials
- 80 mV: sensitive
- 60 mV: less sensitive
- 120 mV: less sensitive
Ewald’s 2\textsuperscript{nd} Law: asymmetry
acceleration / inertia of mass

elasticity

viscosity (friction)

latency SP = 0.8 ms

max. deflection

cupula = 2 ms

maximum deflection ≈ 1°

latency VOR = 8 ms
canals are insensitive for constant rotations

\[
\text{viscosity (friction)} \quad \text{mass} \quad \text{elasticity}
\]

\[
\text{back}_{\text{cupula}} = 20 \text{ s}
\]
canals are insensitive for translations or gravity (specific mass endolymphhe = specific mass cupula)

exceptions: alcohol, cupulolithiasis etc
canals are insensitive for translations or gravity (specific mass endolymph = specific mass cupula). Exceptions: alcohol, cupulolithiasis etc
- increase of sensitivity
- calculation of velocity

duration 20 s ⇒ 60 s

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
velocity storage: mainly for horizontal canals

\[ \text{duration}_{\text{deflection cupula}} = 2 \text{ ms} \]
\[ \text{duration}_{\text{cupula back}} = 20 \text{ s} \]
\[ \text{duration}_{\text{velocity storage}} = 60 \text{ s} \]
\[ \text{duration}_{\text{central adaptation}} > 300 \text{ s} \]
Ewald’s 1st Law: optimal sensitivity
we need 3 dimensions: 3 canals
Ewald’s 2\textsuperscript{nd} Law
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)
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

- $B \sim$ friction / viscosity (max. friction: endolymphe moves with canal)
- $K \sim$ elasticity cupula (no elasticity: cupula does not bend back)
- $I \sim$ endolymphe mass, size (no inertia: no movement)
Theoretical model canal: 2nd order system leads to the following differential equation:

\[ \ddot{q} = \ddot{\Theta} + B \dot{\Theta} + K \Theta \]

- \( q \) - angle head rotation
- \( \Theta \) - angle cupula deflection
- \( I \) - endolymphe mass, size
- \( B \) - friction (viscosity)
- \( K \) - elasticity cupula

Cupula deflection depends on viscosity, elasticity and mass.
frequency dependence canals: gain

- Canal senses acceleration, cupula deflection indicates head velocity.

- $0.1 \text{ Hz} \rightarrow 10 \text{ Hz}$

- Sensitivity

- $B \sim \text{friction (visc)}$

- $K \sim \text{elasticity}$

- $I \sim \text{mass}$
frequency dependence canals: gain

sensitivity

VS

0.1 Hz 10 Hz

calorics chair head impulses

frequency (Hz)
canal senses acceleration, cupula deflection indicates head velocity

frequency dependence canals: phase

\[
\begin{align*}
1 / T_{\text{low}} &= \frac{K}{B} \\
1 / T_{\text{high}} &= \frac{B}{I}
\end{align*}
\]

\( I \sim \text{mass} \)
\( B \sim \text{friction (visc)} \)
\( K \sim \text{elasticity} \)
frequency dependence canals: phase ($\approx$ time constant)

VS

+90°

-90°

0.1 Hz  10 Hz

calorics  chair  head impulses
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

sensitivity

general population

elderly > 65 yo

frequency (Hz)

0.01 Hz 0.1 Hz 10 Hz
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
utriculus
sacculus

medial

lateral

forwards-backwards, sideways translations

forwards-backwards, up and downs translations
theoretical model otolith membrane: again 2\textsuperscript{nd} order system

M $\sim$ otoconia mass

B $\sim$ friction (viscosity)

K $\sim$ elasticity otoconia-membrane
theoretical model otolith membrane: 2\textsuperscript{nd} order system

leads to the following differential equation

\[
(1 - \frac{\rho_e}{\rho_o}) \ddot{X} = \Delta \dot{X} + \frac{B}{M} \Delta \dot{X} + \frac{K}{M} \Delta X
\]

\(\ddot{X}\) acceleration head
\(\Delta X\) relative displacement membrane
\(M\) \sim otoconia mass
\(B\) \sim friction (viscosity)
\(K\) \sim elasticity otolith membrane
\(\rho_e\) and \(\rho_o\) density endolymph \(cq\) otoconia
gain = membrane shift / head acceleration

1 / T_{\text{low}} = \frac{K}{B} \quad 1 / T_{\text{high}} = \frac{B}{I}

I \sim \text{mass}
B \sim \text{friction (visc)}
K \sim \text{elasticity}

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
sensitivity

- 0.2 Hz
- 2 Hz
- 20 Hz

frequency (Hz)

velocity storage network: canal-statolith interaction

statolith

canals

correct        tilt or translation

- 0.2 Hz
- 2 Hz
- 20 Hz
sensitivity

vision and/or proprioception

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
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

<table>
<thead>
<tr>
<th>Orientation in Space</th>
<th>Constant Rotation or Stand Still?</th>
</tr>
</thead>
</table>

### Statoliths

<table>
<thead>
<tr>
<th>Orientation in Space</th>
<th>Constant Translation or Stand Still?</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Orientation Relative to Gravity</th>
<th>Tilt or Translation?</th>
</tr>
</thead>
</table>

**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

motion sickness

age (years)
many hair cells receive efferent input
the brain controls the periphery
canals
utriculus
sacculus
inf
lat
med
sup
vestibular nuclei
lumbal thoracal cervical
cerebellum
neo paleo archi
omn
io
inc
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
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 feedback to keep vestibular function optimal

cerebellum = vestibular adaptive control centre

image stabilisation
balance
spatial orientation
VOR
Vestibulo-Collic, Cervico-Collic, Vestibulo-spinal
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

- dominance right vestibular hemisphere respective side of labyrinth stimulation

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

- efferent projections
thank you for your kind attention
EyeSeeCam® (München)

ICS Impulse® (Sydney)
small VOR correction

head latency
eye

normal VOR

normal dynamic vision
normal head impulse test

poor dynamic vision
abnormal head impulse test

some patients:
covert saccades: sensory substitution

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