the vestibular system: function and dysfunction

Herman Kingma,
- Department of ORL, Maastricht University Medical Centre
- Maastricht Research Institute for Mental Health and Neuroscience, Maastricht University
- Faculty of Biomedical Engineering, Technical University Eindhoven
which complaints are related to vestibular deficits?

which complaints are related to natural limitations?

which complaints are not related to a vestibular problem?
which complaints are related to vestibular deficits?

which complaints are related to natural limitations?

which complaints are not related to a vestibular problem?
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)
permanent complaints / symptoms in patients with centrally compensated vestibular function loss

- increased neuro-vegetative sensitivity
- reduced orientation in space
- hypersensitivity for optokinetic stimuli
- reduced automatisation of balance
- reduced dynamic visual acuity
- secondary: fear/anxiety and fatigue
- gravitoreceptors
  - blood pressure sensors in large blood vessels

- labyrinths

- CNS
  - interpretation
  - learning
  - adaptation
  - compensation

- somatosensory
  - e.g. foot sole pressure

- vision

- hearing

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

- image stabilisation

- spatial orientation

- balance control

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  - e.g. foot sole pressure

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- balance control
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permanent complaints / symptoms in patients with centrally compensated vestibular function loss

- increased neuro-vegetative sensitivity
- reduced automatisation of spatial orientation
- hypersensitivity for optokinetic stimuli
<table>
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<th>Vestibular Impact upon Postural Control</th>
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<td>- Regulation of Muscle Tone Relative to Gravity</td>
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<td>- Regulation of COM Relative to Base of Support Balancing Correction Steps</td>
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<td>- Labyrinths Important for Balance at Low Speed Learning Motor Activities → Automatisation</td>
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visual cortex

hippocampus
basal ganglia
spinal pattern generator

vn
cer
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

slow tandem walk  fast tandem walk
permanent complaints / symptoms in patients with centrally compensated vestibular function loss

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gravity

e.g. foot sole pressure

spatial orientation

somasensory
e.g. foot sole pressure

vision

image stabilisation

balance control
VOR: 8 msec
OKR and Smooth pursuit: >75 msec
head impulse test in unilateral loss
standard video (50 Hz)
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

Normalized VA difference vs Velocity [km/h]

- 0.21
- 0.20
- 0.30

bilateral loss: visual acuity drops below 0.4
which complaints are related to vestibular deficits?

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which complaints are not related to a vestibular problem?
- acute vertigo in case of a sudden change of function of one labyrinth (harmonie vestibulaire)

- severe nausea, imbalance and intolerance to head movements in case of sudden bilateral loss (no nystagmus)

- permanent complaints / symptoms in patients with centrally compensated unilateral or bilateral vestibular function loss
  - increased neuro-vegetative sensitivity
  - orthostatic hypotension
  - reduced automatisation of orientation in space
  - increased sensitivity for optokinetic stimuli
  - reduced automatisation of balance
  - reduced automatisation of gaze stabilisation
  - reduced dynamic visual acuity
  - oscillopsia
  - secondary: fear/anxiety and fatigue
a vestibular function loss implies permanent impairment analogue to hearing and visual losses

examples
- Meniere’s disease when attacks are absent or disappeared
- neuritis vestibularis after central compensation
- bilateral vestibulopathy after central compensation
- vestibular loss schwannoma (also after extirpation)
many vestibular syndromes where vertigo is the leading symptom

- Benign Paroxysmal Positioning Vertigo and Nystagmus
- vestibular neuritis or labyrinthitis / peripheral vestibular ischemia
- pseudo vestibular neuritis: vestibular TIA or infarction
- motion sickness / mal de debarquement
- Meniere’s disease (MD)
- recurrent vestibulopathy (vestibular Meniere? no early stage of MD?)
- vestibular migraine (benign paroxysmal vertigo of childhood?)
- vestibular paroxysms (neuro-vascular compression vestibular nerve, analogon trigeminus neuralgia)
- vestibular epilepsy
- fistula / superior canal dehiscence syndrome (SCDS)
-- central vestibular vertigo
which complaints are related to vestibular deficits?

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clinical relevant knowledge of function and compensation of peripheral lesions
auditory: sounds = high frequency vibrations
vestibular: movements = low frequency vibrations
canals: rotations
statoliths: translations + tilt
myosine filaments

ion channels

80 mV

60 mV

120 mV

action potentials

sensitive

less sensitive
acceleration / inertia of mass → elasticity
viscosity (friction)

maximum deflection ≈ 1°

latency SP = 0.8 ms
max. deflection_{cupula} = 2 ms
latency VOR = 8 ms
Ewald's 1st Law
Ewald’s 2nd Law
Ewald’s 2\textsuperscript{nd} Law

we almost always stimulate 2 labyrinths

asymmetries only 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

right  left

nose
frequency dependence
semicircular canals?
frequency dependence canals: gain

sensitivity

0.1 Hz 10 Hz

frequency (Hz)

calorics chair head impulses
ageing (>60) frequency dependence canals
presbyo-vertigo

sensitivity

general population

everly > 65 yo

frequency (Hz)

0.01 Hz  0.1 Hz  10 Hz
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
The diagram illustrates the anatomy of the inner ear, focusing on the sacculus and utriculus. The sacculus is responsible for detecting up and downs translations, while the utriculus detects forwards-backwards and sideways translations.

Images of both the sacculus and utriculus are shown, with the sacculus measuring 300 µm and the utriculus also measuring 300 µm.
frequency dependence
semicircular statolith systems?
frequency dependence statolith systems (tilt or translation)
limitations labyrinth

canals: no difference between constant rotation and stand still

statoliths: no difference between constant translation and stand still
no difference between tilt and translation
sensitivity

statolith

frequency (Hz)

0.2 Hz

2 Hz

20 Hz
sensitivity

- statolith
- canals

frequency (Hz)

0.2 Hz 2 Hz 20 Hz

correct  tilt or translation
some nasty 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

conclusion: statolith input needs to be confirmed by other senses, otherwise it will be neglected
which complaints are related to natural limitations?

motion sickness!
no pathology: motion sickness
= natural limitation + neuro-vegetative sensitivity

multi-sensory reference stored in the brain based on learning for fast detection of spatial orientation

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multi-sensory input: no movement
detection of gravity vector no problem

detection: no movement

---

comparison
stationary reference

**fast** detection

voluntary self motion

• voluntary control
• feed back
• anticipation

detection of gravity vector no problem
boat = passive motion

- passive movement
- only feedback
- impact upon motor control
- anticipation?

Detection of gravity vector can be a problem

stationary reference

fast detection requires learning

boat movement

boat = passive motion
inadequate stationary reference

• passive movement
• only feed back gravity vector? learned motor patterns do not apply anymore

boat + self motion

problem with selection of right reference for motor activity: motion sickness

built up of new reference and motor learning

fast identification, more automatic but not easy for everybody

before learning

after learning

boat + self motion

anticipation
off shore again ......

wrong reference and adapted to passive motion

no dynamic sensory input

illusion of motion

mal de débarquement

anti-motion sickness drugs: reduce sensitivity and alertness learning versus sedation
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?

motion sickness!

- almost all subjects are susceptible with correct stimulus
  unless a low neuro-vegetative sensitivity
- a (partly) working labyrinth is prerequisite for Motion Sickness:
sensitivity

motion sickness

age (years)
which complaints are related to vestibular deficits?

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“not typical” vestibular

often called aspecific dizziness:

- light headiness
- decrease of consciousness
- moving black spots
- uncertainty, anxiety (unless secondary)
- other
most common causes of aspecific dizziness

- side effect of medication
- fear, anxiety, panic disorders
- hyperventilation
- hypo or hyper tension
- cardio vascular diseases, arythmia
- poorly controlled diabetes
VOR
Vestibulo-Collic, Cervico-Collic, Vestibulo-spinal
VOR
Vestibulo-Collic, Cervico-Collic
Vestibulo-Spinal, Perception
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

vestibular most active hemisphere located in the non-dominant hemisphere

labyrinth stimulation activities ipsi-laterally

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

joint processes set at work to achieve fast and optimal recovery
central compensation: neuroplasticity

- increase of visual sensitivity
- usage of commisural input
- formation of new neurons stimulated by movement and high dosage betahistine (Lacour 2004)
- reprogramming connections to balance spontaneous activity of both vestibular nuclei
harmonie vestibulaire
directed towards lesion side:
- slow phase
- falling
- finger pointing

sedation impairs central compensation
methyl-prednisolon (100 mg every 2-days ▼) + betahistine 2-3 dd. 48 mg
250 mg/ml endolymphe 50 mg/kg in cats

NO cerebellar shut down

new neurons and connections
central vestibular compensation (e.g. Lacour 1992 - 2010)

maximum compensation is reached within 3-12 months
  - stimulated by movement
  - impaired by immobilization and vestibular sedation

→ STATIC components of compensation are good (in rest)
  - neuroplasticity in VN is powerful

→ DYNAMIC components of compensation are poor (during movement)
  - automatization of complex gait and balance remains impaired
  - image stabilisation remains poor
  - hypersensitivity to optokinetic stimuli remains
  - impaired spatial orientation remains
localisation of labyrinth dysfunction in detail is now more often possible, but requires complex equipment.
patients with acquired bilateral vestibular arereflexia

- gentamycine intoxication
- ageing
- auto-immune, vascular

- are they just fine after optimal compensation and sensory substitution?
- how can we quantify residual function
- is there any need for a vestibular implant?
miniature 6-DOF detector in the belt 4x30x40 mm (gyroscopes + accelerometers)

vibration belt on the trunk 12 vibrators

battery + processor with zero-posture reset
belt with vibrators

sensor random  
sensor on

placebo effect?

double blind placebo controlled study
vibrotactile vest and belt: supports body tilt and body rotation perception

- In 64 out of 83 patients the effect is significant more than placebo:
  vibrotactile feedback improves stance and gait considerably in many patients

- 1 system is now used by a patient for about 1 year: he reports impressive increase in his quality of life

- The new adjustable system allows easy testing in patients

- 5 patients are now provided with an individually customized belt for long-term evaluation

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vest  adjustable belt  individual belt
vestibular implants: state of the art knowledge (Guyot, Merfeld, Kingma, Stokroos)
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- partial restorage of VOR with electro stimulation via gyroscopes in animals possible
- good and fast adaptation to chronic sustained stimulation in animals
- Rubinstein: influence Meniere attacks?
- surgical routes in humans explored for stimulation of PC, HC and AC

Geneve: nerve stimulation / Maastricht: intra-ampular stimulation

- per-operative and chronic stimulation studies in humans (Geneva/Maastricht):
  - peroperative 3D stimulation of all 3 canals possible: proof of concept
  - 4 patients with chronic implantation of VI with 1 electrode (PC nerve)
- chronic 2D and 3D implantation studies running now (Geneva/Maastricht)
I hope this was useful to you to understand the function of the vestibular system better.

thank you

hermankingma@gmail.com