

Can we differentiate a “conductive” from a “sensorineural” VOR impairment on vHIT?

Superior canal dehiscence VS acute vestibular loss

A. Castellucci ¹, S. Martellucci ², P. Malara ³, M. Alfarghal ⁴, E. Armato ⁵.

¹ AUSL-IRCCS di Reggio Emilia, Italy, ² AUSL di Latina, Italy, ³ Centromedico, Bellinzona, Switzerland, ⁴ King Abdulaziz Medical City, Jeddah, Saudi Arabia, ⁵ University of Padova, Italy.



Background

The **video-head impulse test (vHIT)** represents an accurate device for the measurement of the semicircular canal **vestibulo-ocular-reflex (VOR)**. Specific **lesion patterns** have been detected in several vestibular disorders **depending on the underlying pathomechanism and etiology**. The **morphology of the slow-phase VOR for the hypoactive canals at the vHIT** has not been analyzed yet. A **phase-shift (or phase-lag) of the VOR** at the vHIT has been generally attributed to **artifacts**.

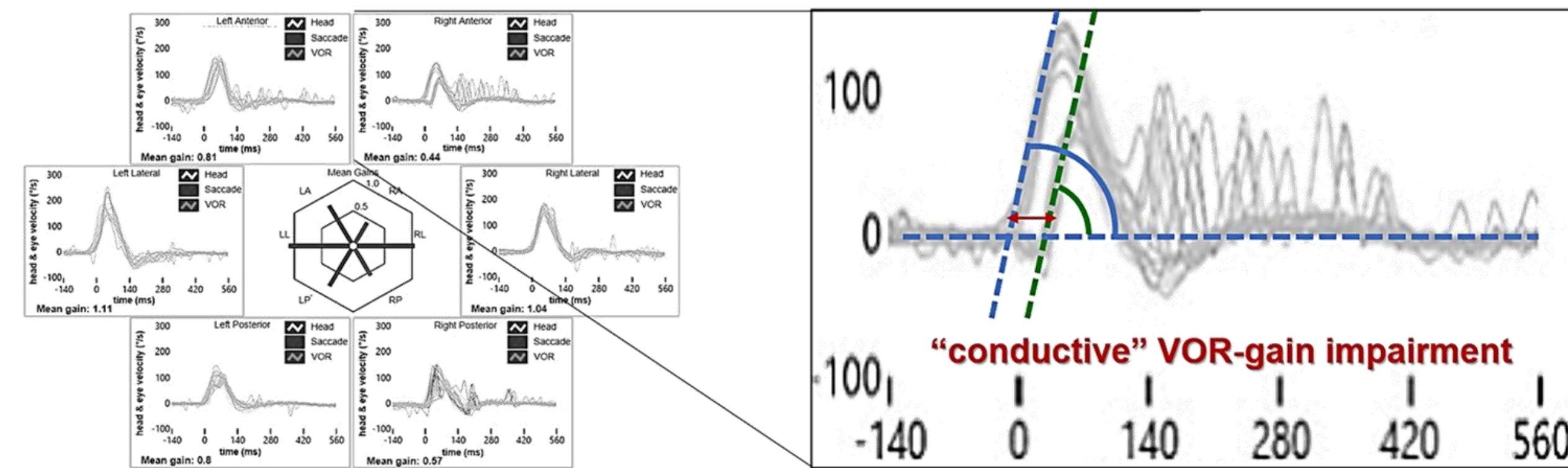
In **acute unilateral vestibulopathy (AUVP)**, a neural inflammation or an ischemic damage of vestibular sensors leads to a **reduced activity of the affected neuroepithelium (“sensorineural” VOR-gain impairment)**. Conversely, **superior canal dehiscence (SCD)** can generate a selective VOR-gain impairment for the dehiscent canal likely through an **endolymphatic flow dissipation** via a low-impedance pathway due to the dehiscence (**“conductive” VOR-gain impairment**), consistent with the **“third-window mechanism” (TWM)**.

Aims

This preliminary study aims to assess the possibility to **distinguish a “conductive” from a “sensorineural” VOR gain impairment depending on the underlying pathomechanisms**, analyzing the morphology of vHIT-traces.

Conclusions

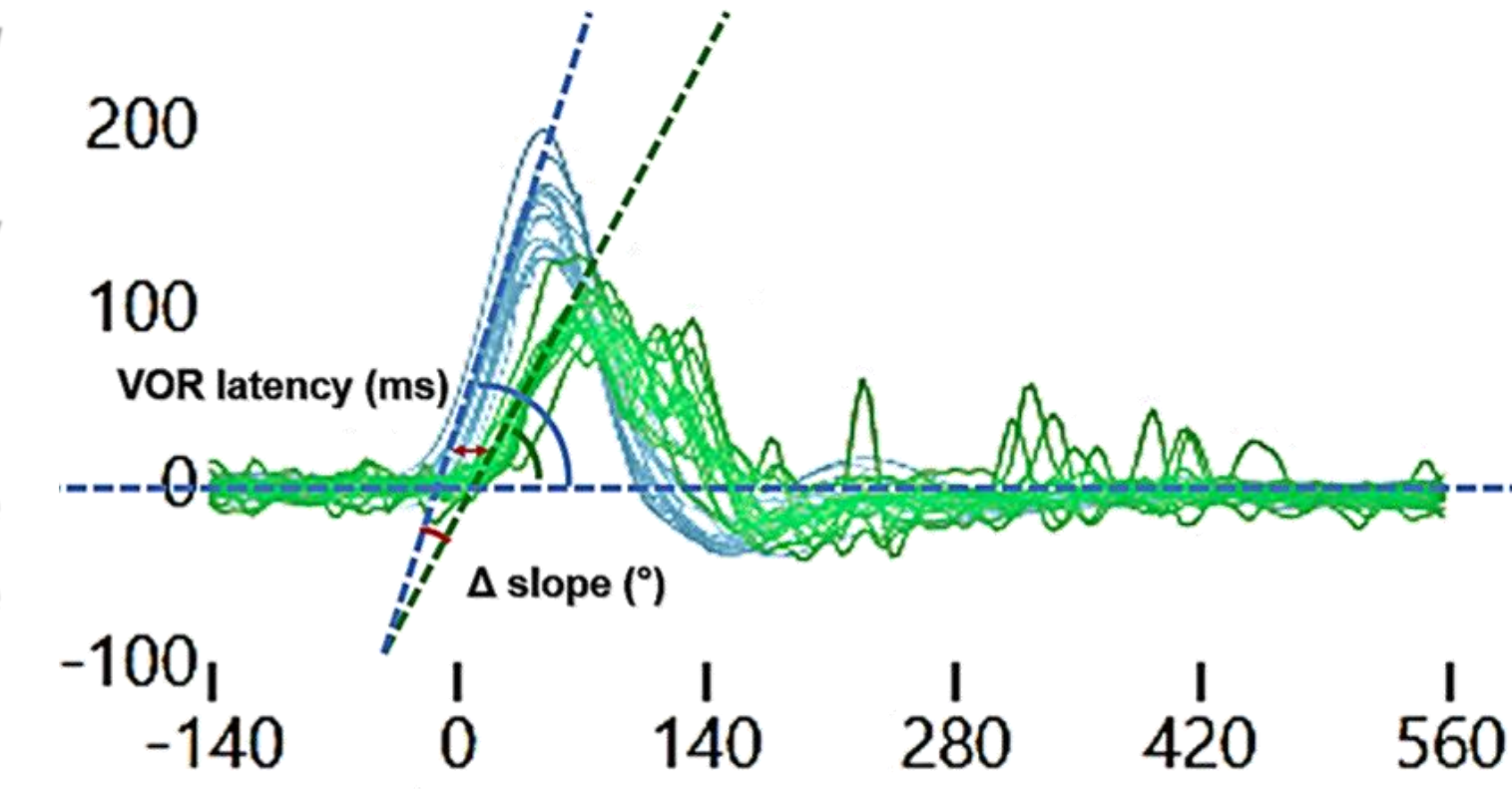
The **different morphologies** of vHIT traces might not be due to artifacts, but rather could give **new insights into the pathomechanisms underlying the VOR impairment**, shedding lights into **new possible applications for the vHIT**.



Further studies with **larger and different cohorts** (including patients with Meniere’s disease, vestibular schwannoma, and other inner ear disorders leading to VOR-gain impairment) **will be needed to confirm our findings**.

Materials and Methods

vHIT data of **10 patients with SCD with TMW symptoms** and VOR-gain impairment for the dehiscent canal and **10 patients with a history of AUVP** exhibiting matched VOR-gains for the affected canals were retrospectively analyzed. **Latency (ms)** and the **difference between the slope of the head trace and the slope of the slow-phase VOR (Δ slope, °)** or the affected and healthy canals were measured and compared.



Results

Canals affected in **SCD** exhibited significantly **higher VOR latencies** and **lower Δ slopes** compared to the affected canals in **AUVP** subgroup, revealing **different pathomechanisms underlying the development of the VOR-gain impairment**.

VOR-latencies of the canals affected in **SCD** were significantly **higher** also compared to **healthy canals**, while **Δ slopes** of the canals affected in **AUVP** were significantly **higher than healthy canals**.

