

Spontaneous Reversing Positional Nystagmus as a sign of simultaneous ampullary and non-ampullary Posterior Canal BPPV

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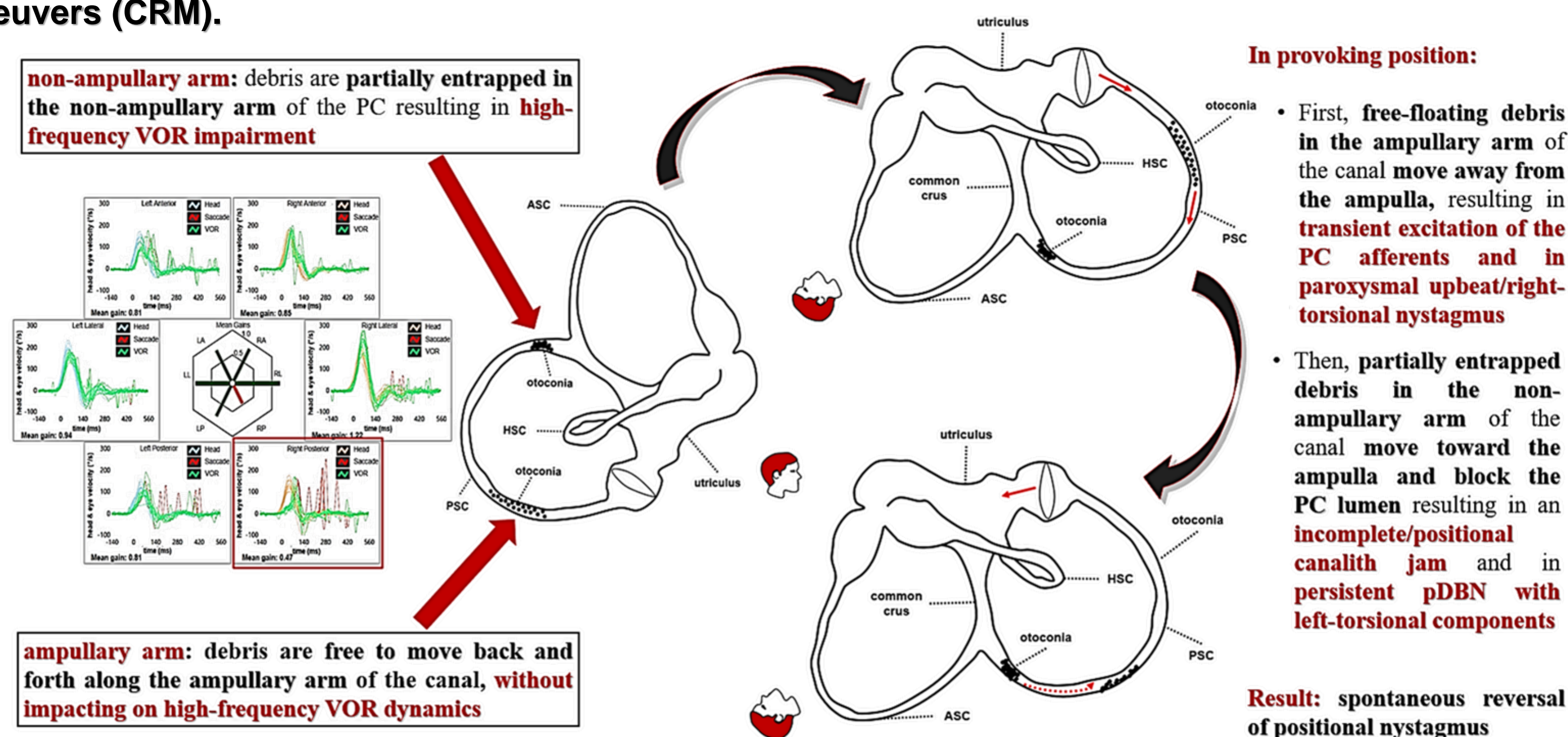
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Background

Reversing positional nystagmus (RPN) represents an atypical positional nystagmus in BPPV as it spontaneously reverses its direction in the same position. The inversion of nystagmus has been related to different pathomechanisms including **rebound cupular deflections**, either **peripheral or central short term adaptation mechanisms of the VOR** and **coexistence of canalolithiasis and cupulolithiasis**. Though it has been mainly described in lateral canal (LC) BPPV, **RPN can also occur in posterior canal (PC) BPPV** when paroxysmal upbeat nystagmus (pUBN) with ipsilesional torsional components is followed by persistent downbeating nystagmus (pDBN) in Dix-Hallpike test (DHT). Recently, **the use of the vHIT has been recommended in the differential diagnosis of BPPV with pDBN** (anterior canal BPPV versus PC-BPPV involving the non-ampullary arm) as it can detect endolymphatic flow dysfunctions due to an **incomplete canalith jam** which alters high-frequency flow dynamics leading to VOR-gain impairment for the affected canal by. This study aims to provide **possible etiopathogenetic hypotheses to explain RPN in PC-BPPV**.

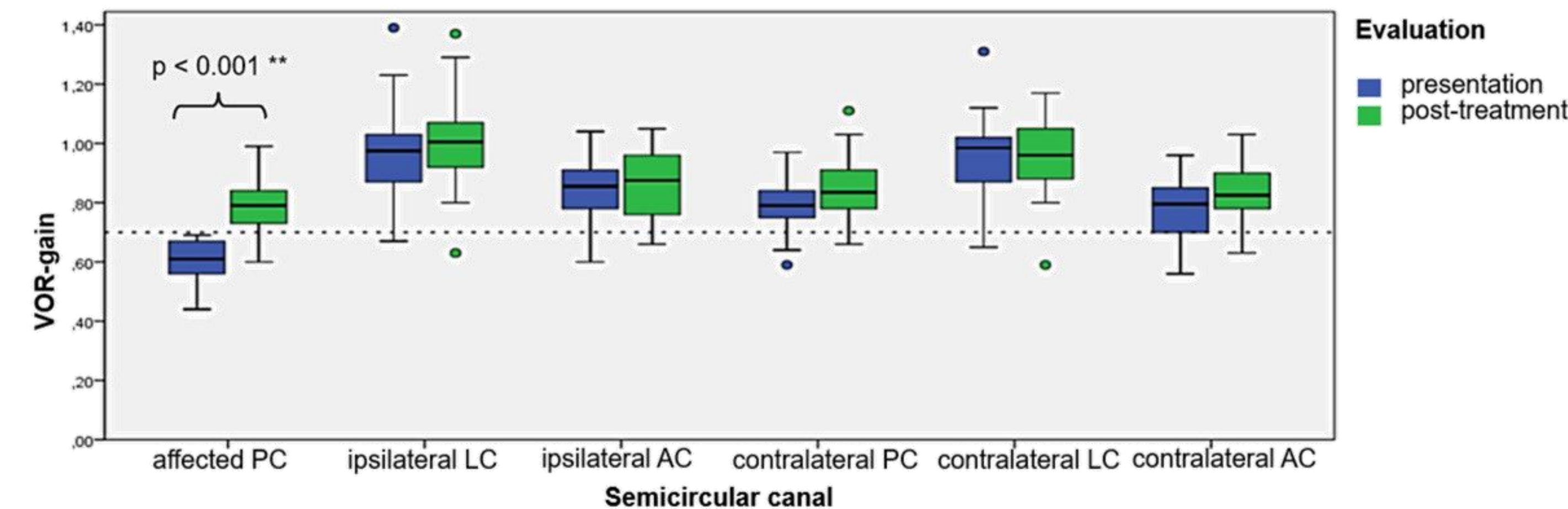
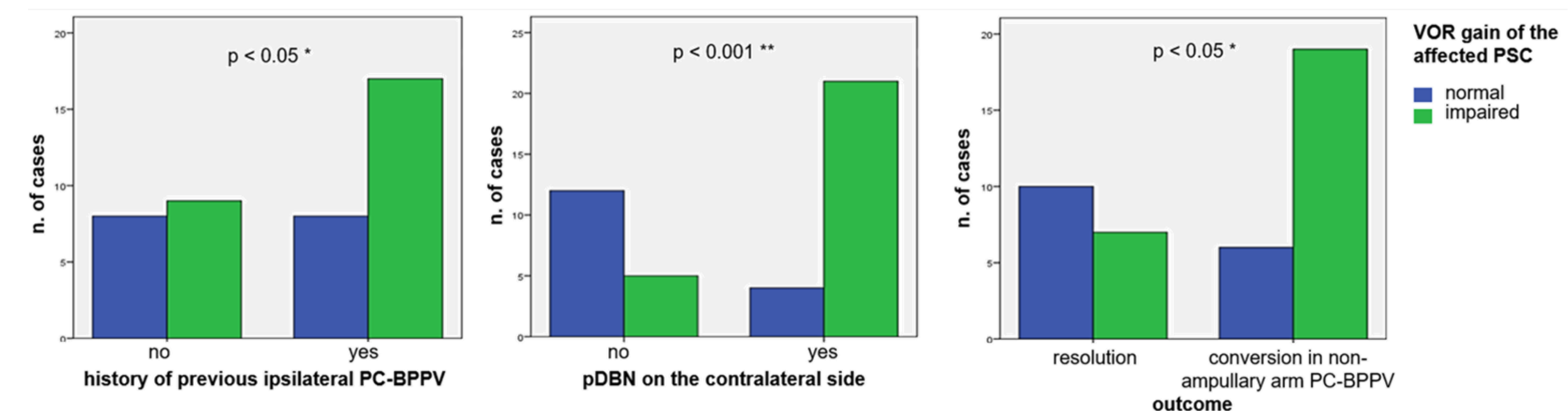
Materials and Methods

The records of **42 patients** (5 males, 37 females, mean age 68.4±11.2 years) **with PC-BPPV exhibiting RPN** were retrospectively reviewed, including **history of previous ipsilateral PC-BPPV**, **nystagmus duration**, presence of **pDBN in contralateral DHT**, canal **VOR-gain values at the vHIT** and the **outcome of canalith repositioning maneuvers (CRM)**.



Results

Patients with reduced PC VOR-gain (62% of cases) exhibited **higher rate of previous ipsilateral PC-BPPV** ($p < 0.05$), **shorter first-phase pUBN** ($p < 0.05$), **higher incidence of pDBN in contralateral DHT** ($p < 0.001$) and **lower success rate of CRM** ($p < 0.05$) compared to the patients with normal PC VOR-gain. **PC-VOR gain normalized in all cases with impaired values after symptoms resolution** ($p < 0.001$).



Conclusions

According to our data, **in a consistent group of patients with PC-BPPV with RPN, the second-phase pDBN** could be likely explained by the **coexistence of an additional clot of debris partially entrapped in the non-ampullary arm of the PC (incomplete canalith jam)**, which results in the detection of pDBN in bilateral DHT and in a slight PC impairment and prevents immediate otolith reposition with CRM.