

CHARACTERIZATION OF THE VESTIBULO-COCHLEAR NERVE MOTION IN VIVO USING A PHASE CONTRAST MRI SEQUENCE

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INTRODUCTION

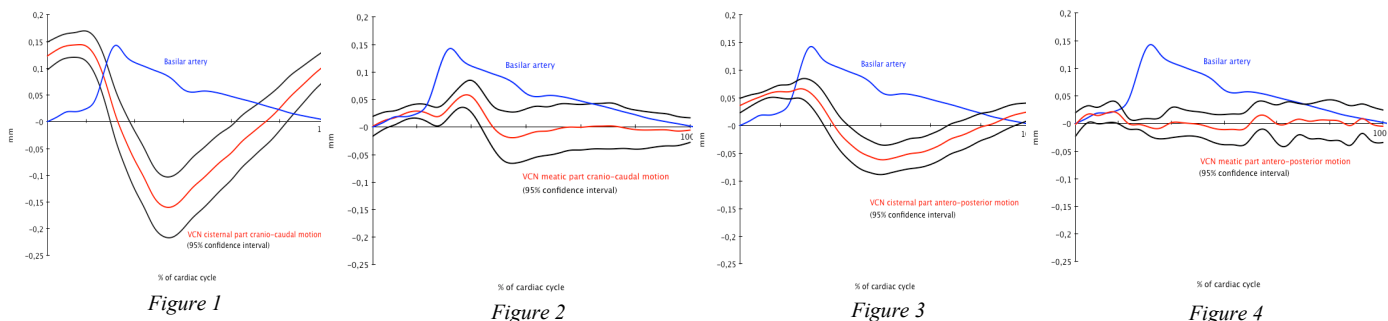
Pulsatile motion of the brain is well known. Transmission of the systolic pulse wave into the cerebral structures leads to systolic expansion and motion of the brain. The amplitude is particularly high at the level of the brain stem¹. No studies have so far investigated cranial nerve motion because of the size of these structures and of technical limitations of MRI systems. The purpose of this study was to qualify and quantify the vestibule-cochlear nerve (VCN) motion at the level of the cerebello-pontine angle. The pathophysiology of diseases like microvascular syndromes affecting nerves in this anatomical region could be better understood.

MATERIALS AND METHODS

This prospective study was performed on a 3 Tesla MRI scanner (Signa HDxt; GE Healthcare, Milwaukee, WI, USA) by using a velocity-encoded cine phase-contrast pulse sequence. Measurements were performed in 28 healthy volunteers, 19 men aged 20-53 years (mean 42 +/- 12.8 years), and 9 women aged 21-64 years (mean 36 +/- 15.7 years). Scanning was approved by the local review board. An ECG gating was used. First the VCN was optimally located by a succession of three 3D FIESTA sequences. T2 sequences (TE 155 ms / TR 2800 ms) followed by phase-contrast sequences in the same planes were prescribed, allowing us to assess the cranio-caudal (CC) and antero-posterior (AP) VCN motion. The velocity encoding parameter (Venc) was 2 cm/sec. To quantify blood flow into the basilar artery, another phase contrast sequence was added to the protocol with a Venc of 80 cm/sec. To correct for constant offset of the static tissue, we hypothesized that tissue would return to its initial position after a cardiac cycle. Quantification of tissue motion relied on the integration of speed along time. A time shift algorithm was used to align brain stem motion waveforms between subjects. Using basilar artery flow waveforms, a systolic phase was defined as the phase of maximum flow velocity, and was used as a reference to realign all subjects' data in time.

RESULTS

Measurements in the seven regions of interest (ROI) were successfully completed in 20 out of 28 subjects. Incomplete measurements were mainly due to anatomical failures. The blood flow pattern of basilar arteries was superimposed to the cranial nerve motion waveforms allowing the definition of a systolic phase (one third of the cardiac cycle) and a diastolic phase (the other two thirds). In the CC direction, the cisternal VCN started a caudal motion in the middle of the systolic phase, which lasted until the first quarter of the diastolic phase, then came back to its starting position at a slower speed (mean amplitude 0.37 +/- 0.14 mm) (Figure 1). The motion of the meatic VCN was similar with a shift in time of 16% of the cardiac cycle, was initially composed by a cranial motion followed by a caudal motion, but its mean amplitude was two times smaller than for the cisternal part of the VCN (0.17 +/- 0.08 mm) (Figure 2). The motion of the pons was similar in shape to the motion of the cisternal VCN (0.28 +/- 0.09 mm). In the AP direction, the cisternal VCN had a posterior motion in the last third of the systolic phase, which lasted until the first quarter of the diastolic phase, then during the rest of the cardiac cycle the cisternal VCN came back to its starting position with a slower speed (0.19 +/- 0.08 mm) (Figure 3). The meatic VCN had smaller motion amplitude (0.16 +/- 0.14 mm), and seemed to oscillate without a clear pattern (Figure 4). The motion of the pons followed the same shape as the cisternal VCN but with smaller amplitude (0.10 +/- 0.03 mm).



DISCUSSION

Our study demonstrates that VCN motion is a cardiac-cycle-dependent movement like brain motion¹. We elaborated a “string oscillated” model of the VCN, attached to the bulbopontine sulcus of the pons and so moving with it, firmly fixed in the temporal bone at its entrance in the cochlea and vestibule. This model can explain the diminution of motion amplitude between cisternal and meatic parts in both directions. The measurements of the CC motion of the pons are similar to those found in literature^{2,3}. There are some limitations to this study. We hypothesized that the longitudinal (left-right) motion would be insignificant during the oscillations. Seven failures consisted in the impossibility to localize the VCN and to draw ROIs accurately; the last was technical consisting in an erroneous heart beat recording. It will be interesting to measure the change of VCN motion in patients presenting a microvascular compression syndrome (tinnitus, vertigo), and see if there would be an alteration in the magnitude of VCN motion relative to the offended vessel.

REFERENCES

1. Enzmann, D.R., Pelc, N.J. Radiology 1992;185:653-660.
2. Maier, S.E., Hardy, C.J., Jolesz, F.A. Radiology 1994;193:477-483.
3. Soellinger, M., Rutz, A.K., Kozerke, S., Boesiger, P. Magn Reson Med 2009;61:153-162.