Journal of Biomechanics 204 (2026) 113354

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech

Short communication

High-Frequency wall vibration correlates with growth of a vertebrobasilar dolichoectatic Aneurysm: A case study

Kei Yamamoto [a,b], Luca Soliveri [c], Michela Bozzetto [c], Chiara Emma Campiglio [d], Andrea Remuzzi [d], Luigi Andrea Lanterna [e], Kristian Valen-Sendstad [a,*]

[a] Department of Computational Physiology, Simula Research Laboratory, Oslo, Norway
[b] Department of Mathematics, University of Oslo, Oslo, Norway
[c] Department of Biomedical Engineering, Istituto di Ricerche Farmacologiche Mario Negri IRCCS, Bergamo, Italy
[d] Department of Management, Information and Production Engineering, University of Bergamo, Bergamo, Italy
[e] Depatment of Neurosurgery, ASST Papa Giovanni XXIII Hospital, Bergamo, Italy

ARTICLE INFO

Keywords:
Dolichoectasia
Basilar artery
Fluid–structure interaction
Vascular wall vibration
Case study

ABSTRACT

Vertebrobasilar dolichoectatic aneurysms (VBDAs) are a rare cerebrovascular condition with poorly understood pathophysiology. While complex flow-induced stresses are believed to influence aneurysm initiation, progression, and rupture, previous computational VBDA or more broadly cerebral aneurysm studies have focused primarily on time-averaged wall shear stress at a single time point. In this study, we investigate a broader range of mechanical stimuli using longitudinal data to explore their association with VBDA growth and remodeling, which may lead to its rupture.

We performed high-fidelity/resolution fluid–structure interaction simulations of the posterior circulation in a patient with VBDA using medical images from three time points. We computed stresses at and within the wall, with the latter decomposed into low-frequency (pulsations) and high-frequency (vibrations) components. We examined whether pulsations, vibrations, or conventional hemodynamic metrics independently contributed to VBDA growth.

At the first time point, inflow jets from the left and right vertebral arteries collided, creating local flow instabilities and associated wall vibrations above the vertebrobasilar junction. Within this region, spectrograms, showing the evolution of vibration frequency, revealed several narrow band vibration peaks between 50 and 150 Hz. Only regions of high vibration amplitude correlated with VBDA growth; conventional hemodynamic metrics and pulsations showed no such association. These results were broadly consistent across the second and third time point, during which further growth of the VBDA was confirmed.

This case study demonstrates a qualitative spatial–temporal association between flow-induced high-frequency wall vibrations and growth of the VBDA, supporting the hypothesis that wall vibrations may act as a mechanobiological stimulus.

1. Introduction

Vertebrobasilar dolichoectatic aneurysms (VBDAs) are rare vascular diseases characterized by elongation (dolicho) and dilatation (ectasia) of cerebral arteries (Pico et al., 2015). The rarity of the disease and the lack of clear anatomical definition make diagnosis challenging. Similarly, the absence of well-defined treatment criteria complicates surgical planning (Del Brutto et al., 2017). Although several endovascular procedures have shown promising outcomes (Boardman and Byrne, 1998; Wu et al., 2013), craniotomy for VDBA remains challenging due to complex surgical access to the basilar artery, with higher complication rates than saccular aneurysms in the Circle of Willis (Dobrocky et al., 2021).

Furthermore, the pathophysiology of aneurysm formation and progression in general remains unclear (Krings et al., 2011), with several hypotheses suggesting genetic (Finney et al., 1976), immunological (Goldstein et al., 2010), or mechanobiological origins (Hoi et al., 2008). In particular, the latter has gained attention due to increased evidence demonstrating the role of wall shear stress (WSS) both in homeostasis

* Corresponding author at: Simula Research Laboratory, Norway
E-mail address: kvs@simula.no (K. Valen-Sendstad).

https://doi.org/10.1016/j.jbiomech.2026.113354
Accepted 6 May 2026
Available online 7 May 2026

and adverse vascular remodeling (Turjman et al., 2014). While previous in vitro studies demonstrated the degenerative role of disturbed flow on endothelial cells (Chien, 2007), histological studies specific to VBDAs indicated disruption of elastic tissue in the tunica media (Caplan, 2005) and neointimal hyperplasia (Nakatomi et al., 2000) rather than endothelial dysfunction. We then wondered whether, to explain VBDA growth, research should be extended beyond WSS induced effects on the endothelial cells, to also consider potential adverse effects of mechanical stresses acting within the entire vascular wall.

This idea is not new–clinicians and researchers have long investigated a range of vascular phenomena (Ferguson, 1970; Simkins and Stehbens, 1974). In 1970, Ferguson, who reported turbulence in aneurysms, hypothesized that flow-induced vibrations may cause degenerative change in vascular tissue. However, the lack of suitable experimental or computational tools at the time may have made it difficult to quantitatively measure or model these phenomena. Only recently, our high-fidelity fluid–structure interaction (FSI) simulations revealed transitional flow-induced high-frequency wall vibrations (Souche and Valen-Sendstad, 2022; Bruneau et al., 2023), consistent with Ferguson's early predictions. While we hypothesized that these vibrations may act as a mechanobiological stimulus, rather than a source of mechanical fatigue as originally proposed by Ferguson, our earlier work was limited to a single time point. This reflects standard clinical imaging protocols aimed at minimizing patient risk, which restricted the ability to assess the relationship between wall vibrations and aneurysm progression. Consequently, the aim of this study was to investigate a broader range of mechanical stimuli beyond WSS metrics alone, specifically high-frequency vessel wall vibrations, and their potential association with aneurysm growth using retrospective longitudinal data.

2. Methods

A 59-year-old male patient with thunderclap headache and hypertension was examined at the Department of Neurosurgery, ASST Papa Giovanni XXIII Hospital (Bergamo, Italy). Informed consent for the use of the patient's clinical data was obtained from the next of kin following the patient's death. The local institutional review board determined that this retrospective study is exempt from the approval requirements. The

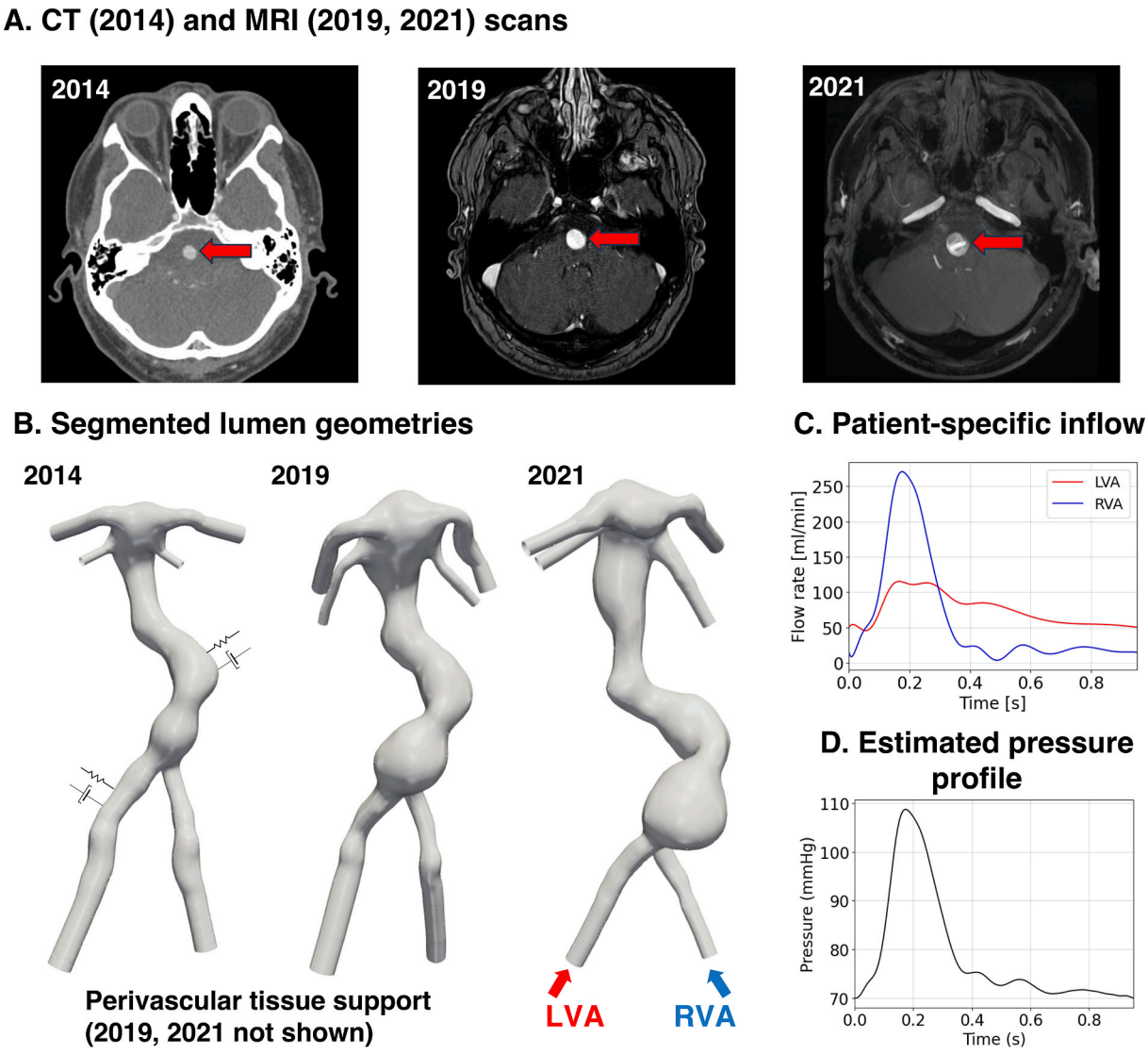

Fig. 1. A. Axial cut of computed tomography (CT) and magnetic resonance image (MRI) scans with red arrows indicating the vertebrobasilar dolichoectatic aneurysm and its growth over time. B. Lumen geometries segmented from medical images for three time points, comprising the left and right vertebral arteries (LVA/RVA), the basilar artery, and the superior and posterior cerebral arteries. The spring-damper system represents the perivascular tissue with viscoelastic damping effect. C. Patient-specific waveform measured in 2021 using phase-contrast MRI. D. Estimated pulsatile blood pressure waveform applied at the vascular wall. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

neurological examination in 2014 revealed VBDA, as shown in Fig. 1A, with a mean diameter of 6.7 mm, exceeding the threshold for ectasia of 4.5 mm (Smoker et al., 1986). Due to high risk of complications associated with surgery, close monitoring was chosen. In 2019, a follow-up examination revealed further growth of the VBDA with a mean diameter reaching 8 mm; however, a conservative approach with continued monitoring was still preferred. Subsequent follow-up in 2021 detected further aggravation with symptomatic mass effect and surgery was decided due to high risk of aneurysm rupture. While the surgery was successful, the patient passed away following the development of ischemic lesions. The mean diameter in 2021 was 7.4 mm, slightly lower than in 2019 potentially due to stenosis progression in certain regions, but the length and tortuosity of the basilar artery increased from 59 mm to 73 mm and from 0.19 to 0.40, respectively, indicating elongation. A histogram illustrating the change of basilar artery radii is given in the Supplementary Material (Fig. S1).

We performed high-fidelity FSI simulations using VBDA geometries (Fig. 1B) reconstructed from CT (2014) and MRI (2019, 2021) images. The segmented models were visually verified by the neurosurgeon who performed the intervention to ensure consistency across the imaging modalities. Patient-specific waveforms (Fig. 1C), derived in 2021 using phase contrast MRI, were imposed with time-averaged flow rate of 78 mL/min and 68 mL/min at the inlet of left and right vertebral artery, respectively. These values are within physiologically plausible ranges for vertebral arteries (Ford et al., 2005; Amin-Hanjani et al., 2015). A pulsatile blood pressure was estimated (Fig. 1D) and applied to the luminal side of the wall, allowing the pressure to vary between 70 to 110 mmHg. Viscoelastic damping from the perivascular environment was incorporated in FSI simulations (Anagnostakou et al., 2022), via the spring-damper system (Fig. 1B), with increased damping for the 2021 case to reflect the symptomatic mass effect. Simulations and post-processing were performed using the methods described in Yamamoto et al. (2025b) and the open-source software VaSP (Yamamoto et al., 2025a). Fluid velocity and wall displacement were decomposed using a 25 Hz cutoff frequency (Khan et al., 2017) to identify flow instabilities, and to distinguish between wall pulsations versus vibrations, respectively.

3. Results

Qualitative results are summarized in Fig. 2. Focusing first on 2014, inflow jets from the left and right vertebral arteries collided, creating flow instabilities in the basilar artery (Fig. 2A) and in regions near the superior cerebellar and posterior cerebral branches. The flow instabilities induced wall vibrations (B) with peak time-averaged amplitudes of 0.2 μm in the vertebrobasilar junction and less pronounced vibrations in the distal basilar artery. Within these two regions, wall spectrograms (C), representing the evolution of vibration frequency, revealed narrowband frequencies around 50 and 150 Hz, whereas much weaker vibrations were observed at the left vertebral artery. Qualitative comparison of the morphology from 2014 to 2019 suggests a spatial association between wall vibration and VBDA growth.

Despite morphological changes, results from 2019 were broadly similar to those of 2014 with stronger vibration amplitude near the distal end of the basilar artery. The wall spectrogram from the vertebrobasilar junction exhibited a more prominent narrowband frequency at 150 Hz compared to 2014. Comparison of the morphology from 2019 to 2021 indicates a continued qualitative spatial association between wall vibration and VBDA growth. The 2021 case exhibited similar patterns of flow instabilities to the previous two time points, but vibration amplitude was markedly reduced, which we hypothesize may be due to increased compression from the perivascular environment associated with the observed symptomatic mass effect.

Finally, we computed conventional hemodynamic metrics, time-averaged wall shear stress (TAWSS) and oscillatory shear index (OSI), and time-averaged low-frequency displacement (pulsation), stress, and strain. However, none showed a qualitative spatial–temporal association with aneurysm growth. Details are provided in the Supplementary Material (Fig. S3).

4. Discussion

This study focused on a VBDA patient with longitudinal medical imaging. Although a larger cohort is required for generalization, we observed a qualitative spatial–temporal association between flow-induced high-frequency wall vibrations and VBDA progression in 2014 and 2019, when growth was confirmed. As neither conventional hemodynamic indices nor traditional low-frequency wall displacement (pulsation), stress, or strain showed a qualitative spatial–temporal association with growth, our findings suggest that vascular wall vibration may act as an adverse mechanobiological stimulus despite the limited number of time points.

That being said, given the rarity of the disease, our study, like many others (Hassan et al., 2004; Graziano et al., 2013), focused on a single case. However, a notable exception is the study by Brinjikji et al. (2018), who conducted CFD simulations on a large cohort of 37 unruptured VBDAs followed longitudinally. By comparing hemodynamic factors, they showed that unstable aneurysms tended to exhibit low WSS and high OSI, driven by complex vortical flow structures. We similarly found that such flow patterns, here quantified as flow instabilities above 25 Hz, can induce high-frequency wall vibrations. However, unlike the broad distribution of OSI (high-sensitivity), we observed that these vibrations are more localized to the region of wall remodeling (high-specificity). This finding does not contradict the conclusions of Brinjikji et al., as complex vortices would likely lead to elevated wall motion, potentially with high-frequency components if their study had focused on high-fidelity modeling of the vascular wall. As such, our results appear complementary, although we point to a different mechanism of vascular remodeling.

In fact, elevated OSI has been reported to correlate not only with VBDA growth but also with aneurysm rupture status in the Circle of Willis (Can and Du, 2016; Liang et al., 2019). Although OSI is an incomplete descriptor of unstable flow (Peiffer et al., 2013, Gallo et al., 2016, Khan et al., 2017), elevated OSI may reflect underlying flow instabilities, and potentially wall vibrations, if these had been properly resolved in space and time (Valen-Sendstad and Steinman, 2014). This interpretation is consistent with transitional flow observed in many bifurcation aneurysms (Khan et al., 2021) and aligns with recent studies showing that spatial (Rezaeitaleshmahalleh et al., 2025) and temporal (Fukuda et al., 2025) gradients of WSS correlate more strongly with rupture status than conventional time-averaged metrics. The phenomenon appears not to be limited to cerebral aneurysms (Souche and Valen-Sendstad, 2022; Bruneau et al., 2023), as flow-induced wall vibrations have also been reported in malfunctioning arteriovenous fistulas (Soliveri et al., 2024; Bozzetto et al., 2024; Soliveri et al., 2025). While the pathophysiology and cellular composition of each vascular wall may differ, it is reasonable to hypothesize that high-frequency wall vibrations elicit similar mechanobiological effects. Supporting this, several in vitro studies have investigated the impact of vibrations on vascular tissue. In 1963, Roach (1963) observed experimentally the coexistence of poststenotic dilatation and turbulence, where the presence of the latter was inferred from thrill and bruit. Roach hypothesized that flow-induced vibrations could weaken the links between collagen fibers and increase the distensibility of the arterial wall. Around the same time and motivated by the growing clinical concern over the hand-arm vibration syndrome, Ljung et al. (1975) conducted experiments in which isolated rabbit portal vein and thoracic aorta were exposed to vibrations of different frequencies. The results indicated a reduction in active force of smooth muscle cells when vibration frequency exceeded 25 Hz. While these two studies focused on the macroscopic and tissue-level effects of vibrations, Bittle (1994) later proposed a cellular-level hypothesis: that inertial forces from acceleration induce smooth

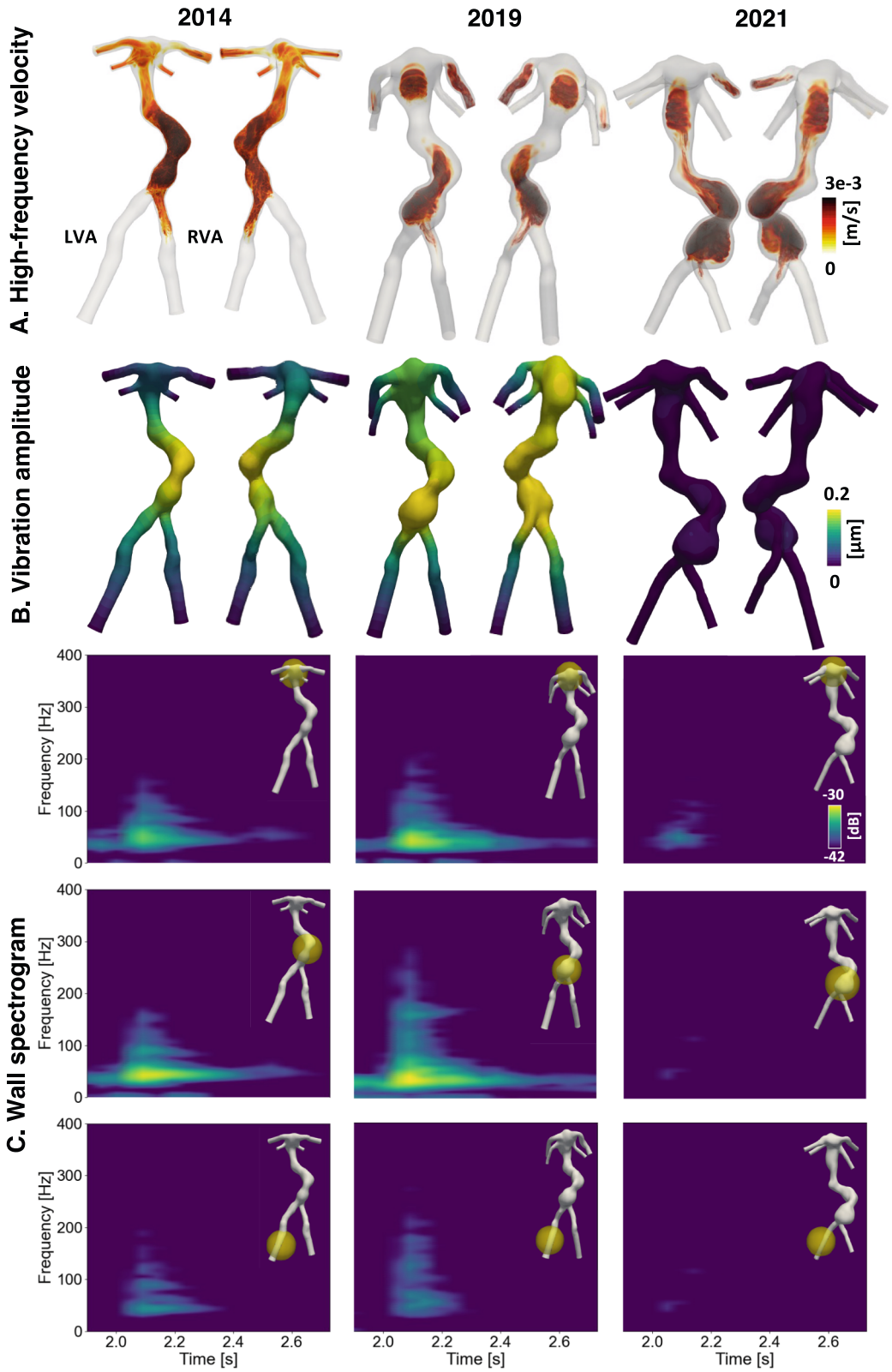

Fig. 2. A. High-frequency flow instabilities (>25 Hz) at peak systole. B. Higher time-averaged wall vibration amplitudes were observed at the distal basilar artery and the vertebrobasilar junction, with the latter showing peak values in 2014 and 2019. C. Wall spectrograms were generated from three different regions, as indicated by yellow spheres, exhibiting narrowband frequency between 50 to 150 Hz within the vertebrobasilar junction for 2014 and 2019. LVA/RVA = left/right vertebral artery. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

muscle cell fatigue by causing asynchronous internal and external motion. Her experiments demonstrated increased cell proliferation in response to vibrations with an amplitude of 12 μm and a frequency of 45 Hz. While prior findings suggest that flow-induced arterial wall vibrations may serve as mechanobiological stimuli promoting vascular growth, the underlying molecular mechanisms of this adverse remodeling remain largely unknown (Carrara et al., 2024). Studying these processes in situ is challenging, as abnormal WSS and flow-induced vibrations often coincide both spatially and temporally. Yet, there is strong support for a distinct role of vibrations, especially in aneurysms, where focal remodeling occurs despite most lacking the intact endothelium needed to mediate WSS-driven changes (Frösen et al., 2004). Taken together, it is plausible that intravascular mechanical stimuli could contribute to vascular remodeling—analogous to externally applied vibration therapies widely investigated in medical science, such as bone regeneration (Thompson et al., 2014) and vibration-induced cancer cell disruption (Ayala-Orozco et al., 2024) at specific frequencies.

However, we acknowledge that our reported frequencies and amplitudes are subject to uncertainties and model limitations (although the vibration location is robust). For example, we assumed wall thickness and stiffness based on a previous experimental study (Robertson et al., 2015), although VBDAs likely exhibit heterogeneous wall properties. Nonetheless, our previous sensitivity study has shown that wall vibrations are robust to those unknown parameters (Yamamoto et al., 2025b), and we expect the impact of wall heterogeneity to be secondary, as the high-frequency vibrations are primarily driven by the unstable flow rather than local wall properties. An additional limitation is that the inflow waveform measured in 2021 was imposed for all three geometries. Temporal changes in patient-specific inflow conditions were therefore not captured and may have influenced the predicted vibration amplitude and spectral content. The vibration frequencies reported here and in our previous aneurysm wall vibration studies (Souche and Valen-Sendstad, 2022; Bruneau et al., 2023) closely match the audible bruits reported clinically (Kosugi et al., 1983; Kurokawa et al., 1994). While the origin of the sound was debated, it was hypothesized to be wall vibrations generating “musical tones” (Aaslid et al., 1982), a mechanism for which Bruneau et al. (2023) recently presented a mechanistic explanation. Given our finding that wall vibration correlates with aneurysm growth, this reinforces the potential of acoustic measurements not only as a non-invasive detection method, but also as a tool for monitoring VBDA growth.

5. Conclusion

This longitudinal single-case study suggests that flow-induced high-frequency wall vibrations may be a possible mechanobiological contributor to VBDA growth, potentially reflecting a broader phenomenon in which pathological vessels sense and remodel in response to such mechanical stimuli.

6. Declaration of generative AI and AI-assisted technologies in the manuscript preparation process.

During the preparation of this work, the authors used ChatGPT to check grammar, clarity, and coherence. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

CRediT authorship contribution statement

Kei Yamamoto: Writing – original draft, Visualization, Software, Methodology. **Luca Soliveri:** Writing – review & editing, Methodology, Formal analysis, Data curation. **Michela Bozzetto:** Writing – review & editing, Data curation, Conceptualization. **Chiara Emma Campiglio:** Writing – review & editing, Funding acquisition. **Andrea Remuzzi:** Writing – review & editing, Conceptualization. **Luigi Andrea Lanterna:** Writing – review & editing, Resources. **Kristian Valen-Sendstad:** Writing – review & editing, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

This study was supported by the EU Horizon 2020 SimCardioTest project (101016496) and the Nepios no profit society. L.S. acknowledges a research fellowship received from Fondazione Dompè. K.Y, K.V.-S, and C.E.C. acknowledge the support from the EU NextGenerationEU (MUR-FondoPromozioneeSviluppo-DM737/2021). The simulations were performed on resources provided by Sigma2 - the National Infrastructure for High-Performance Computing and Data Storage in Norway, grant number nn9249k.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbiomech.2026.113354.

References

Aaslid, R., Markwalder, T.M., Nornes, H., 1982. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J. Neurosurg. 57 (6), 769–774. https://doi.org/10.3171/jns.1982.57.6.0769.

Amin-Hanjani, S., Du, X., Pandey, D.K., Thulborn, K.R., Charbel, F.T., 2015. Effect of age and vascular anatomy on blood flow in major cerebral vessels. J. Cereb. Blood Flow Metab. 35 (2), 312–318. https://doi.org/10.1038/jcbfm.2014.203.

Anagnostakou, V., Epshtein, M., Ughi, G.J., King, R.M., Valavanis, A., Puri, A.S., Gounis, M.J., 2022. Transvascular in vivo microscopy of the subarachnoid space. Journal of NeuroInterventional Surgery 14 (5), 420–428. https://doi.org/10.1136/neurintsurg-2021-018544.

Ayala-Orozco, C., Galvez-Aranda, D., Corona, A., Seminario, J.M., Rangel, R., Myers, J. N., Tour, J.M., 2024. Molecular jackhammers eradicate cancer cells by vibronic-driven action. Nat. Chem. 16 (3), 456–465. https://doi.org/10.1038/s41557-023-01383-y.

Bittle, B. B. (1994). An investigation into the role of arterial wall vibration in the pathogenesis of atherosclerosis.

Boardman, P., Byrne, J.V., 1998. Giant fusiform basilar artery aneurysm: Endovascular treatment by flow reversal in the basilar artery. Br. J. Radiol. 71 (MAR.), 332–335. https://doi.org/10.1259/bjr.71.843.9616247.

Bozzetto, M., Remuzzi, A., Valen-Sendstad, K., 2024. Flow-induced high frequency vascular wall vibrations in an arteriovenous fistula: a specific stimulus for stenosis development? Phys. Eng. Sci. Med. 47 (1), 187–197. https://doi.org/10.1007/s13246-023-01355-z.

Brinjikji, W., Chung, B., Yong-Hong, D., Wald, J.T., Mut, F., Kadirvel, R., Kallmes, D.F., Rouchaud, A., Lanzino, G., Cebral, J.R., 2018. Hemodynamic characteristics of stable and unstable vertebrobasilar dolichoectatic and fusiform aneurysms. Journal of NeuroInterventional Surgery 10 (11), 1102–1107. https://doi.org/10.1136/neurintsurg-2018-013756.

Bruneau, D.A., Steinman, D.A., Valen-Sendstad, K., 2023. Understanding intracranial aneurysm sounds via high-fidelity fluid-structure-interaction modelling. Communications Medicine 3 (1), 1–11. https://doi.org/10.1038/s43856-023-00396-5.

Can, A., Du, R., 2016. Association of hemodynamic factors with intracranial aneurysm formation and rupture: Systematic review and meta-analysis. Neurosurgery 78 (4), 510–519. https://doi.org/10.1227/NEU.0000000000001083.

Carrara, E., Soliveri, L., Poloni, S., Bozzetto, M., Campiglio, C.E., 2024. Effects of high-frequency mechanical stimuli on flow related vascular cell biology. International Journal of Artificial Organs, August. https://doi.org/10.1177/03913988241268105.

Caplan, L.R., 2005. Dilatative arteriopathy (dolichoectasia): what is known and not known. Ann. Neurol. 57 (4), 469–471. https://doi.org/10.1002/ana.20447.

Chien, S., 2007. Mechanotransduction and endothelial cell homeostasis: the wisdom of the cell. Am. J. Physiol. Heart Circ. Physiol. 292 (3). https://doi.org/10.1152/ajpheart.01047.2006.

Del Brutto, V.J., Ortiz, J.G., Biller, J., 2017. Intracranial arterial dolichoectasia. In Frontiers in Neurology. Issue JUL). Frontiers Media s.a. 8. https://doi.org/10.3389/fneur.2017.00344.

Dobrocky, T., Piechowiak, E.I., Goldberg, J., Barvulsky Aleman, E., Nicholson, P., Lynch, J., Bervini, D., Kaesmacher, J., Agid, R., Krings, T., Raabe, A., Gralla, J., Pereira, V.M., Mordasini, P., 2021. Absence of pontine perforators in vertebrobasilar dolichoectasia on ultra-high resolution cone-beam computed tomography. Journal of

NeuroInterventional Surgery 13 (6), 580–584. https://doi.org/10.1136/neurintsurg-2020-016818.
Ferguson, G.G., 1970. Turbulence in human intracranial saccular aneurysms. J. Neurosurg. 33 (5), 485–497. https://doi.org/10.3171/jns.1970.33.5.0485.
Finney, H.L., Roberts, T.S., Anderson, R.E., 1976. Giant intracranial aneurysm associated with Marfan's syndrome. J. Neurosurg. 45 (3), 342–347. https://doi.org/10.3171/jns.1976.45.3.0342.
Ford, M.D., Alperin, N., Sung, H.L., Holdsworth, D.W., Steinman, D.A., 2005. Characterization of volumetric flow rate waveforms in the normal internal carotid and vertebral arteries. Physiol. Meas. 26 (4), 477–488. https://doi.org/10.1088/0967-3334/26/4/013.
Frösen, J., Piippo, A., Paetau, A., Kangasniemi, M., Niemelä, M., Hernesniemi, J., Jääskeläinen, J., 2004. Remodeling of saccular cerebral artery aneurysm wall is associated with rupture: Histological analysis of 24 unruptured and 42 ruptured cases. Stroke 35 (10), 2287–2293. https://doi.org/10.1161/01.STR.0000140636.30204.da.
Fukuda, S., Shimogonya, Y., & Watanabe, A. (2025). Two possible hemodynamic mechanisms underlying the growth of cerebral aneurysms depending on their size: The NHO CFD ABO study. https://doi.org/10.1177/0271678X251325972.
Gallo, D., Steinman, D.A., Morbiducci, U., 2016. Insights into the co-localization of magnitude-based versus direction-based indicators of disturbed shear at the carotid bifurcation. J. Biomech. 49 (12), 2413–2419. https://doi.org/10.1016/j.jbiomech.2016.02.010.
Goldstein, D.A., Timpone, J., Cupps, T.R., 2010. HIV-associated intracranial aneurysmal vasculopathy in adults. J. Rheumatol. 37 (2), 226–233. https://doi.org/10.3899/jrheum.090643.
Graziano, F., Russo, V.M., Wang, W., Khismatullin, D., Ulm, A.J., 2013. 3D Computational Fluid Dynamics of a Treated Vertebrobasilar Giant Aneurysm: a Multistage Analysis. Am. J. Neuroradiol. 34 (7), 1387–1394. https://doi.org/10.3174/ajnr.a3373.
Hassan, T., Ezura, M., Timofeev, E.V., Tominaga, T., Saito, T., Takahashi, A., Takayama, K., Yoshimoto, T., 2004. Computational simulation of Therapeutic Parent Artery Occlusion to Treat Giant Vertebrobasilar Aneurysm. Am. J. Neuroradiol. 25 (1), 63–68.
Hoi, Y., Gao, L., Tremmel, M., Paluch, R.A., Siddiqui, A.H., Meng, H., Mocco, J., 2008. In vivo assessment of rapid cerebrovascular morphological adaptation following acute blood flow increase: Laboratory investigation. J. Neurosurg. 109 (6), 1141–1147. https://doi.org/10.3171/JNS.2008.109.12.1141.
Khan, M.O., Chnafa, C., Gallo, D., Molinari, F., Morbiducci, U., Steinman, D.A., Valen-Sendstad, K., 2017. On the quantification and visualization of transient periodic instabilities in pulsatile flows. J. Biomech. 52, 179–182. https://doi.org/10.1016/j.jbiomech.2016.12.037.
Khan, M.O., Toro Arana, V., Najafi, M., MacDonald, D.E., Natarajan, T., Valen-Sendstad, K., Steinman, D.A., 2021. On the prevalence of flow instabilities from high-fidelity computational fluid dynamics of intracranial bifurcation aneurysms. J. Biomech. 127, 110683. https://doi.org/10.1016/j.jbiomech.2021.110683.
Kosugi, Y., Goto, T., Ikebe, J., Johshita, H., Takakura, K., 1983. Sonic detection of intracranial aneurysm and AVM. Stroke 14 (1), 37–42. https://doi.org/10.1161/01.STR.14.1.37.
Krings, T., Mandell, D.M., Kiehl, T.R., Geibprasert, S., Tymianski, M., Alvarez, H., TerBrugge, K.G., Hans, F.J., 2011. Intracranial aneurysms: from vessel wall pathology to therapeutic approach. Nat. Rev. Neurol. 7 (10), 547–559. https://doi.org/10.1038/nrneurol.2011.136.
Kurokawa, Y., Abiko, S., Watanabe, K., 1994. Noninvasive detection of intracranial vascular lesions by recording blood flow sounds. Stroke 25 (2), 397–402. https://doi.org/10.1161/01.STR.25.2.397.
Liang, L., Steinman, D.A., Brina, O., Chnafa, C., Cancelliere, N.M., Pereira, V.M., 2019. Towards the Clinical utility of CFD for assessment of intracranial aneurysm rupture - a systematic review and novel parameter-ranking tool. Journal of NeuroInterventional Surgery 11 (2), 153–158. https://doi.org/10.1136/neurintsurg-2018-014246.
Ljung, B., Sivertsson, R., 1975. Vibration-Induced Inhibition of Vascular Smooth Muscle Contraction. J. Vasc. Res. 12 (1), 38–52. https://doi.org/10.1159/000158037.
Nakatomi, H., Segawa, H., Kurata, A., Shiokawa, Y., Nagata, K., Kamiyama, H., Ueki, K., Kirino, T., 2000. Clinicopathological study of intracranial fusiform and dolichoectatic aneurysms: Insight on the mechanism of growth. Stroke 31 (4), 896–900. https://doi.org/10.1161/01.STR.31.4.896.
Pico, F., Labreuche, J., Amarenco, P., 2015. Pathophysiology, Presentation, Prognosis, and Management of Intracranial Arterial Dolichoectasia. in the Lancet Neurology Vol. 14(8, 833–845. doi:10.1016/S1474-4422(15)00089-7.
Peiffer, V., Sherwin, S.J., Weinberg, P.D., 2013. Computation in the rabbit aorta of a new metric - the transverse wall shear stress - to quantify the multidirectional character of disturbed blood flow. J. Biomech. 46 (15), 2651–2658. https://doi.org/10.1016/j.jbiomech.2013.08.003.
Rezaeitaleshmahalleh, M., Lyu, Z., Mu, N., Nainamalai, V., Tang, J., Gemmete, J.J., Pandey, A.S., Jiang, J., 2025. Improving Prediction of Intracranial Aneurysm Rupture Status using Temporal Velocity-Informatics. Ann. Biomed. Eng. 53 (4), 1024–1041. https://doi.org/10.1007/s10439-025-03686-2.
Roach, M.R., 1963. Changes in arterial distensibility as a cause of poststenotic dilatation. Am. J. Cardiol. 12 (6), 802–815. https://doi.org/10.1016/0002-9149(63)90284-4.
Robertson, A.M., Duan, X., Aziz, K.M., Hill, M.R., Watkins, S.C., Cebral, J.R., 2015. Diversity in the Strength and Structure of Unruptured Cerebral Aneurysms. Ann. Biomed. Eng. 43 (7), 1502–1515. https://doi.org/10.1007/s10439-015-1252-4.
Simkins, T.E., Stehbens, W.E., 1974. Vibrations recorded from the adventitial surface of experimental aneurysms and arteriovenous fistulas. Vasc. Surg. 8 (3), 153–165. http://www.ncbi.nlm.nih.gov/pubmed/4841073.
Smoker, W.R.K., Price, M.J., Keyes, W.D., Corbett, J.J., Gentry, L.R., 1986. High-resolution computed tomography of the basilar artery: 1. Normal size and position. AJNR Am. J. Neuroradiol. 7 (1), 55–60. http://www.ncbi.nlm.nih.gov/pubmed/3082144.
Soliveri, L., Bruneau, D., Ring, J., Bozzetto, M., Remuzzi, A., Valen-Sendstad, K., 2024. Toward a physiological model of vascular wall vibrations in the arteriovenous fistula. Biomech. Model. Mechanobiol. 0123456789. https://doi.org/10.1007/s10237-024-01865-z.
Soliveri, L., Poloni, S., Brambilla, P., Caroli, A., Remuzzi, A., Bozzetto, M., Valen-Sendstad, K., 2025. High-Frequency Vessel Wall Vibrations associate with Stenosis Formation and Arteriovenous Fistula failure. Kidney Medicine 7 (3), 100957. https://doi.org/10.1016/j.xkme.2024.100957.
Souche, A., Valen-Sendstad, K., 2022. High-fidelity fluid structure interaction simulations of turbulent-like aneurysm flows reveals high-frequency narrowband wall vibrations: a stimulus of mechanobiological relevance? J. Biomech. 145, 111369. https://doi.org/10.1016/j.jbiomech.2022.111369.
Thompson, W.R., Yen, S.S., Rubin, J., 2014. Vibration therapy: Clinical applications in bone. Curr. Opin. Endocrinol. Diabetes Obes. 21 (6), 447–453. https://doi.org/10.1097/MED.0000000000000111.
Turjman, A.S., Turjman, F., Edelman, E.R., 2014. Role of fluid dynamics and inflammation in intracranial aneurysm formation. Circulation 129 (3), 373–382. https://doi.org/10.1161/CIRCULATIONAHA.113.001444.
Valen-Sendstad, K., Steinman, D.A., 2014. Mind the gap: Impact of computational fluid dynamics solution strategy on prediction of intracranial aneurysm hemodynamics and rupture status indicators. Am. J. Neuroradiol. 35 (3), 536–543. https://doi.org/10.3174/ajnr.A3793.
Wu, X., Xu, Y., Hong, B., Zhao, W.-Y., Huang, Q.-H., Liu, J.-M., 2013. Endovascular Reconstruction for Treatment of Vertebrobasilar Dolichoectasia: Long-Term Outcomes. Am. J. Neuroradiol. 34 (3), 583–588. https://doi.org/10.3174/ajnr.A3248.
Yamamoto, K., Bruneau, D.A., Ring, J., Dokken, J.S., Valen-Sendstad, K., 2025a. VaSP: Vascular Fluid–Structure Interaction Pipeline. SoftwareX 32, 102392. https://doi.org/10.1016/j.softx.2025.102392.
Yamamoto, K., Bruneau, D.A., Dokken, J.S., Steinman, D.A., Valen-Sendstad, K., 2025b. Impact of Wall Property and Flow Rate Assumptions on Simulations of Flow-Induced Vibration of Intracranial Aneurysms. International Journal for Numerical Methods in Biomedical Engineering 41 (10). https://doi.org/10.1002/cnm.70101.