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College *	College of Liberal Arts and Sciences
Department *	Health and Human Physiology
Title of Research *	Functional and structural vascular responses to increased blood pressure variability is frequency dependent
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Introduction & Purpose *	Enhanced blood pressure variability (BPV), experienced by patients with impaired baroreflex function, leads to cardiovascular end-organ damage even in the absence of hypertension. Different frequency components of BPV correspond to various cardiovascular control mechanisms. We tested the hypothesis the BPV-induced cardiovascular damage depends on the pattern of BPV, such that very low frequency (VLF) BPV, which is enhanced in patients with impaired baroreflex function, is more detrimental than low frequency (LF) BPV.
Experimental Design *	VLF (30s period) or LF (3s period) BPV was induced in normotensive rats by cardiac pacing. During a pacing period of 3 weeks, structural and functional parameters of the long posterior ciliary artery (LPCA) of the iris were assessed using a novel and innovative in vivo imaging technique using a slit lamp biomicroscope. Cardiac function was assessed by echocardiography. Cardiac hypertrophy was assessed by measuring final ventricular weight.
Results *	Preliminary data of this ongoing study demonstrate that after 3 weeks wall-to-lumen ratio tended to increase in rats exposed to VLF BPV, but tended to decrease in rats exposed to LF BPV and in control rats. Endothelial-mediated dilation of the LPCA in response to corneal application of pilocarpine tended to be diminished in rats exposed to VLF BP, while rats exposed to LF BPV had preserved endothelial function. VLF BPV tended to result in a greater degree of cardiac hypertrophy and had diminished ejection fraction when compared to LF BPV or control rats.
Conclusions *	Our results suggest that increased VLF BPV in patients with impaired baroreflex function (e.g., diabetes or hypertension) may contribute to their overall increased cardiovascular risk. Novel treatment strategies aimed at preventing BPV-induced cardiovascular end-organ damage should be based on blood pressure control mechanisms corresponding to VLF BPV (e.g., Renin-Angiotensin-System, catecholamines, nitric-

oxide, myogenic vascular function).

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