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**Ivan Ramirez-Zuniga\*** (ivr3@pitt.edu), **Renee Brady**, **Charles Puelz**, **Kamila Larripa**, **Elisabeth Bangsgaard** and **Mette S. Olufsen**. *The interaction between inflammation and cardiovascular dynamics.*

The acute inflammatory response triggered by infection, trauma or surgery is a delicate system aimed at promoting healing and restoring homeostasis. However, inflammation can result in undesirable effects, such as tissue damage or changes in blood pressure and cardiovascular dynamics. It has been shown that the inflammatory response alters the levels of nitric oxide, a vasodilator. Here, we present a coupled model of the inflammatory response and the cardiovascular system, with nitric oxide as the connecting link. Our model was calibrated to experimental data that was obtained measuring pro-inflammatory mediators (IL-6, CXCL8, and TNF) and the anti-inflammatory cytokine IL-10, in addition to blood pressure and heart rate, over 8 hours in 20 healthy men, given a low dose of lipopolysaccharide (LPS), an endotoxin stimulating inflammation. (Received September 13, 2016)