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Cellular and isolated vessel experiments have shown that pulsatile and laminar shear stress to the endothelium produces significant release of mediators into the circulation. Periodic acceleration (pG(z)) applied to the whole body in the direction of the spinal axis adds pulses to the circulation, thereby increasing pulsatile and shear stress to the endothelium that should also cause release of mediators into the circulation. The purpose of this study was to determine whether addition of pulses to the circulation through pG(z) would be sufficient to increase shear stress in whole animals and to acutely release mediators and how such a physical maneuver might affect coagulation factors. Randomized control experiments were performed on anesthetized, supine piglets. The treatment group (pG(z)) (n = 12) received pG(z) with a motion platform that moved them repetitively head to foot at +/-0.4 g at 180 cpm for 60 min. The control group (n = 6) was secured to the platform but remained on conventional ventilation throughout the 4-h protocol. Compared with control animals and baseline, pulsatile stress produced significant increases of serum nitrite, prostacyclin, PGE(2), and tissue plasminogen activator antigen and activity, as well as D-dimer. There were no significant changes in epinephrine, norepinephrine, cortisol, and coagulation factors between groups or from baseline values. Pulsatile and laminar shear stress to the endothelium induced by pG(z) safely produces increases of vasoactive and fibrinolytic activity. pG(z) has potential to achieve mediator-related benefits from the actions of nitric oxide and prostaglandins.