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Periodic acceleration (pGz), a novel method of ventilatory support, is achieved using a platform that moves cyclically in the headward-footward direction. PGz has been shown to increase vascular shear stress and regional blood flows, as well as decrease pulmonary and systemic vascular resistances. PGz also increases nitric oxide (NO) production. This study was undertaken to determine the effects of pGz on the NO inhibiting effects of N-w-nitro-L-arginine (L-NAME) in vivo, and to determine if increased NO production due to pGz could be reproduced in vitro with isolated arteries. Pigs were assigned to conventional ventilation (CV), or pGz, with no additional breathing assistance. L-NAME was infused in cumulative doses of 1, 3, 10, 30, and 100 mg/kg. Cardiac output decreased in both groups by 50%. There was also a dose-dependent increase in blood pressure, pulmonary artery pressure, and vascular resistances. However, pGz attenuated the vascular response of L-NAME. Isolated porcine aortas exposed to nonpulsatile, pulsatile, and pulsatile flow plus pGz exhibited an increase in nitrites with the addition of pulsatile flow (300%, relative to steady flow), and a further increase with pGz (1000%, relative to steady flow). It has been determined that pGz, a novel method of increasing shear stress on the vascular endothelium. attenuates the vasoactive response to L-NAME. The in vitro experiments demonstrated that increases in NO production in vivo could be reproduced in vitro, which provides the opportunity to investigate the mechanisms of cardiovascular pGz effects.