Abstract

Cardiac disease exacerbation is associated with short-term exposure to vehicular emissions. Diesel exhaust (DE) might impair cardiac performance in part through perturbing efferent sympathetic and parasympathetic **autonomic nervous system** (ANS) input to the heart. We hypothesized that acute changes in ANS balance mediate decreased cardiac performance upon DE inhalation. Young adult heart failure-prone rats were implanted with radiotelemeters to measure heart rate (HR), HR variability (HRV), blood pressure (BP), core body temperature, and pre-ejection period (PEP, a contractility index). Animals pretreated with sympathetic antagonist (atenolol), parasympathetic antagonist (atropine), or saline were exposed to DE (500 μg/m(3) fine particulate matter, 4h) or filtered air and then treadmill exercise challenged. At 1 day postexposure, separate rats were catheterized for left ventricular pressure (LVP), contractility, and lusitropy and assessed for **autonomic** influence using the sympathoagonist dobutamine and surgical vagotomy. During DE exposure, atenolol inhibited increases in HR, BP, and contractility, but not body temperature, suggesting a role for sympathetic dominance. During treadmill recovery at 4h post-DE exposure, HR and HRV indicated parasympathetic dominance in saline- and atenolol-pretreated groups that atropine inhibited. Conversely, at treadmill recovery 21h post-DE exposure, HRV and PEP indicated sympathetic dominance and subsequently diminished contractility that only atenolol inhibited. LVP at 1 day postexposure indicated that DE impaired contractility and lusitropy while abolishing parasympathetic-regulated cardiac responses to dobutamine. This is the first evidence that air pollutant inhalation both causes time-dependent oscillations between sympathetic and parasympathetic dominance and decreases cardiac performance via aberrant sympathetic dominance.