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/m3 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 sympatheoanorexist 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.