Abstract

Epidemiologic studies strongly link short-term exposures to vehicular traffic and particulate matter (PM) air pollution with adverse cardiovascular events, especially in those with preexisting cardiovascular disease. Diesel engine exhaust (DE) is a key contributor to urban ambient PM and gaseous pollutants. To determine the role of gaseous and particulate components in DE cardiotoxicity, we examined the effects of one 4-hour inhalation of whole DE (wDE; target PM concentration: 500 $\mu g/m^3$) or particle-free filtered DE (fDE) on cardiovascular physiology and a range of markers of cardiopulmonary injury in hypertensive heart failure-prone rats. Arterial blood pressure (BP), electrocardiography (ECG), and heart rate variability (HRV, an index of autonomic balance) were monitored. Both fDE and wDE decreased BP and prolonged PR interval during exposure, with more effects from fDE, which additionally increased HRV triangular index and decreased T-wave amplitude. fDE increased QTc interval immediately after exposure, increased atrioventricular (AV) block Mobitz II arrhythmias shortly thereafter, and increased serum high-density lipoprotein 1 day later. wDE increased BP and decreased HRV root mean square of successive differences (RMSSD) immediately post-exposure. fDE and wDE decreased heart rate during the 4th hour of post-exposure. Thus, DE gases slowed AV conduction and ventricular repolarization, decreased BP, increased HRV, and subsequently provoked arrhythmias, collectively suggesting parasympathetic activation; conversely, brief BP and HRV changes after exposure to particle-containing DE indicated a transient sympathetic excitation. Our findings suggest that whole and particle-free DE differentially alter cardiovascular and autonomic physiology and may potentially increase risk through divergent pathways.