Electrocardiographic changes during inhalation exposure to diluted Diesel engine emissions in a rat model of myocardial infarction (MI)

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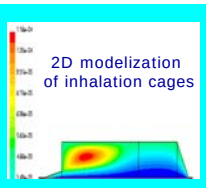
Introduction

To improve understanding of mechanisms involved in increased daily mortality and short term increases in air pollution, we developed and tested a model for investigating the cardiac effects of inhaled diluted Diesel engine emissions as a representative surrogate for urban atmospheric pollution.

Methods

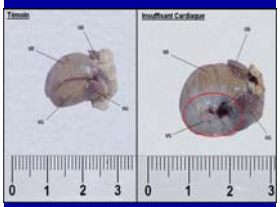
Diesel engine exhausts are drawn directly from the exhaust line, diluted (1:50) with ba double loop at constant pressure and delivered to specifically designed whole body inhalation cages for 3 hour exposure periods.

Chronic heart failure was induced by coronary artery ligation. MI rats were used at least two months after surgery. ECG was continuously monitored and recorded by telemetry for arrhythmia quantitation

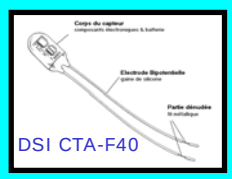


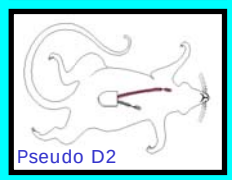
Emission sampling and diluting systems

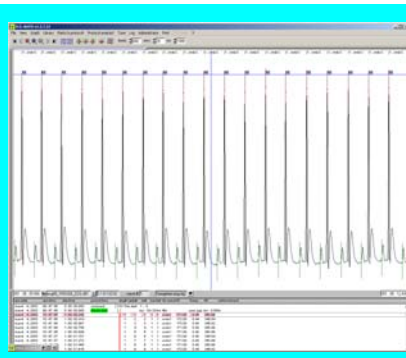
Morphology rat hearts



MI rat Heart Hypertrophy



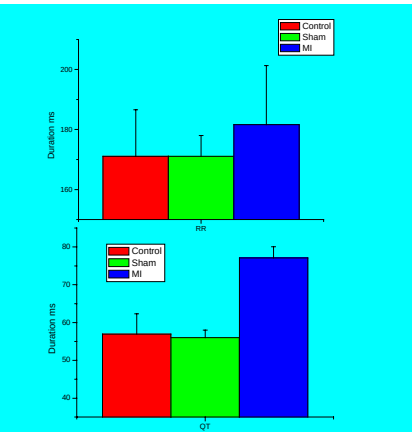




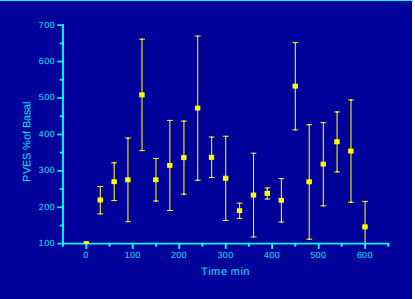
ECG recording (DSI) and automated analysis (EMKA EcgAuto Software)

Results

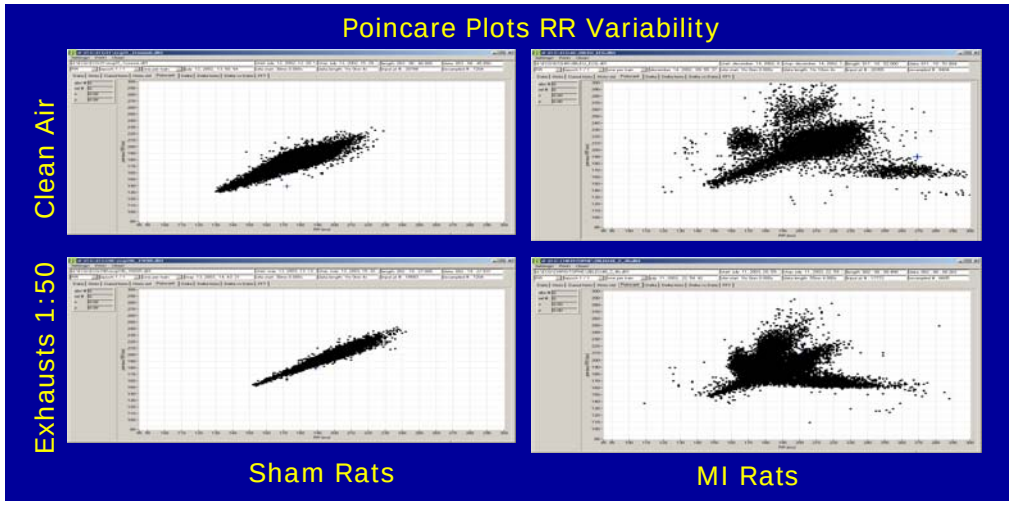
Coronary artery ligation induce decreased heart rate, increased QT interval duration and polymorphic ventricular extrasystolesextrasystoles. Under exhaust exposure, no arrhytmia occurs and a slight decrease in heart rate variability may occur in non MI rats. In MI rats, a marked increase in polymorphic ventricular extrasystole (PVES) occurrence is seen within 15-20 minutes of exposure. After two hours, episodes of unsustained tachycardia are observed in MI rats. 3-5 hours after exposure termination, episodes of sustained ventricular tachycardia occur. 24 hours after exposure termination almost total reversion of exhaust induced changes is seen.



Impact of MI on RR and QT Intervals



Poincare Plots RR Variability



In Conclusion, MI rats show greater sensitivity to engine emissions than non MI rats. Increased PVES frequency is a very early response to exhaust exposure whixh is quqtained for a few hours even upon exposure withdrawal. Apparent decreased heart rate variability (parameter of bad pronistic value) should nbe further studied and quantified. No apparent alteration of QT interval has been observed throughout this study.

This model looks very promising for the study of the mechanisms involved in the cardiac response to combustion aerosol exposure.