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EFFECTS OF WHOLE BODY VIBRATION ON PULMONARY FUNCTION IN RATS

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ABSTRACT

Exposure to vibration stress occurs in numerous occupational settings (e.g. construction, agriculture, mining) where an increase in pulmonary disease has been observed. The objective of this study was to determine if vibration stress alters pulmonary function. Rats (N=10), housed in individual cylindrical chambers, received sinusoidal vibration in a horizontal plane (head to tail) for 4 hrs. at a frequency of 5 Hz and a peak acceleration of 0.75 G. Control rats (N=10) were housed in similar chambers but were not subjected to vibration. During each exposure period, animals received HEPA filtered, conditioned air. Breathing rates and PenH were measured prior to exposure, immediately following exposure, and 18 hrs. post exposure. Respiratory input impedance was measured (SCIREQ, flexiVent) prior to and during a methacholine challenge for anesthetized animals at 18 hrs. post exposure. Although there were no significant differences in baseline respiratory input impedance, animals receiving whole body vibration were hyporesponsive to methacholine at 18 hrs. post exposure.

MATERIALS AND METHODS

Rats were confined in cylindrical chambers and received whole body vibration in a direction normal to the surface of the diaphragm (head to tail). A diagram of the exposure system is shown in Figure 1. The animals were exposed for 4 hrs to a 5 Hz sinusoidal vibration having a maximum acceleration of 0.75 G's (N=10). Control animals were housed in similar chambers for equal time periods but received no vibration (N=10). Breathing rates [1] and PenH (Enhanced Pause) [2] were measured pre-exposure, immediately post-exposure and 18 hrs post-exposure. A flexiVent (SCIREQ) system [3] was used to measure baseline airway resistance and the methacholine response of anesthetized control and exposed animals at 18 hrs post-exposure. Values of respiratory system resistance (R) and elastance (E) were estimated using a sinusoidal perturbation method. The analysis gives the best fit of a single compartment model to measured

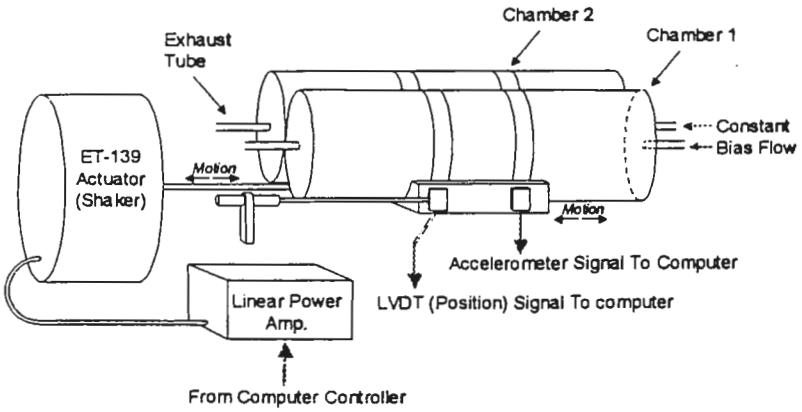

Figure 1. Diagram of system used to vibrate animals in the x-y (head to tail) plane.

data recorded at a perturbation frequency of 1 Hz for 1 second. The model is described by the relationship:

$$Pao(t) = EV + RdV/dt + PO$$

where Pao(t) is the pressure at the airway opening, V is lung volume and PO is a constant.

Control and exposed animals were anesthetized with sodium pentobarbital (83 mg/Kg). Their tracheae were cannulated and attached to the flexiVent system. The animals were ventilated for 5 minutes, then their baseline respiratory system resistance was measured. Following a 5 minute equilibration period, the animals were exposed for 30 seconds to a saline aerosol. This was followed by a series of equilibration periods and exposures to increasing concentrations of methacholine aerosol. The concentration of methacholine was increased until the resistance of the respiratory system had doubled the baseline resistance measurement.

RESULTS & DISCUSSION

Results showed that the breathing rates of animals receiving whole body vibration were not significantly different than the breathing rates of control animals pre-exposure, post-exposure, or 18 hrs. post-exposure. In addition, there were no differences in PenH between control animals and animals receiving whole body vibration measured pre-exposure, post-exposure, or 18 hrs post-exposure. Respiratory system resistance measurements, made 18 hours post exposure, of control animals and animals receiving whole body vibration, however, were significantly different ($P<0.05$) during a methacholine challenge (Figure 2). Control animals responded to lower levels of methacholine than animals receiving whole body vibration even though significant differences in baseline respiratory system resistance measurements following exposure to saline could not be demonstrated.

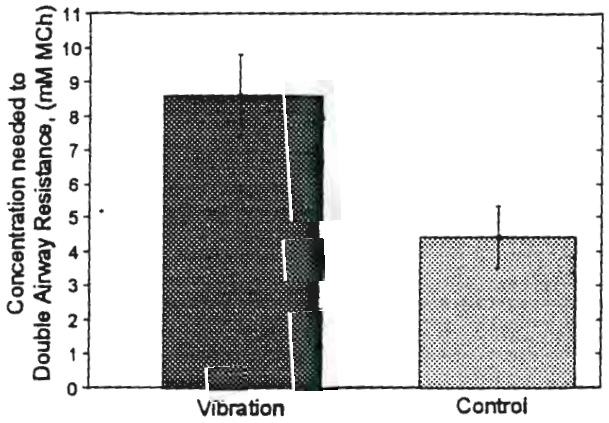

Figure 2. Concentration of methacholine at which the resistance of the respiratory system doubled.

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