

ACUTE EFFECTS OF VIBRATION ON RAT-TAIL NERVES

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Introduction

Hand arm vibration syndrome (HAVS) affects industrial workers exposed to long term hand-transmitted vibration from powered-tools. Peripheral neuropathy is a major component of the symptom complex of HAVS. Long term exposure to vibration causes myelin damage in peripheral nerves and reduces nerve conduction velocities in rats¹. This study addresses the effects of acute vibration at constant acceleration of 49 m/s^2 on myelinated fibers in peripheral nerves in Sprague-Dawley male rats using the 'rat-tail vibration model,' which simulates hand-transmitted vibration².

Methods

Male Sprague-Dawley rats (~300 g) were assigned to vibration groups: 1 hr continuous vibration at 60 Hz; 4 hr continuous exposure at frequencies of 30, 60, 120 or 800 Hz; immediate and 24 hr following a 4-hr cumulative exposure of continuous and intermittent vibration at 60 Hz. Unanesthetized rats were restrained in cages on a nonvibrating platform with their tails placed on a vibrating stage accelerated by a B&K motor type 4809 and vibrated. Intermittent vibration was delivered in bouts of 10 min vibration alternating with 5 min rest periods repeated over 6 hr. Sham controls were restrained without vibration. After vibration exposure, the rats were anaesthetized, and the ventral nerve trunks from the proximal tail segment 7 were fixed in glutaraldehyde, embedded in epon-araldite and sectioned at $0.5 \mu\text{m}$ thickness and stained with toluidine-blue for morphological quantitative analysis. The total number of myelinated axons in each cross-section of the nerve was counted using the Image J software. Myelin damage was identified by focal increase in area and intensity of toluidine-blue staining and unraveling of the myelin sheath. Statistical analysis for comparing sham and the different vibration groups was done using Dunnett's test. Animal treatment and all surgical procedures were approved by the institutional review board and compiled with the Laboratory Animal Welfare Act.

Results

The rats tolerated continuous vibration very well and exhibited no behavioral signs of stress. When exposed to intermittent vibration, there was increased vocalization, a startle reflex at the beginning of each bout of vibration, deposition of porphyrin around the eyes and transient hypersensitivity to touch.

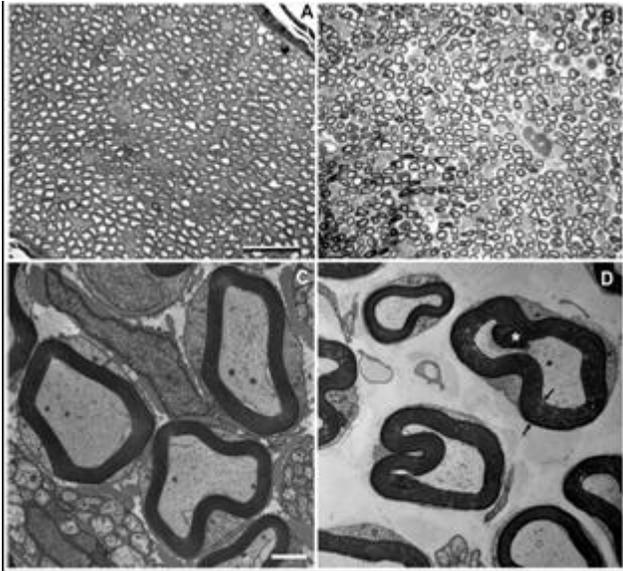


Fig 1: A. The semithin cross section of the tail nerve from a sham control rat demonstrates that the myelin is evenly stained with toluidine blue. B. When vibrated, the myelin stains darker and exhibits focal thickening. C. At the electron microscopic level, the myelin membranes are compact, except for tiny foci of separation in the sham-vibrated control nerves. D. Vibrated nerves exhibit larger and more extensive areas of separation of the myelin membranes (arrows), and frequently the myelin sheaths show decompaction (*). Bar in A equals 40 μm for A, B. Bar in C equals 0.5 μm for C, D.

Table 1: There was an average of 1187 ± 50 myelinated axons in the ventral tail nerve at the level of segment 7. The numbers of myelinated fibers showing delamination are expressed as % of total fibers \pm SEM. All vibration groups were significantly different from the sham vibrated, $*p < 0.05$. CI- Continuous immediate, CS- Continuous 24 hr survival, II- Intermittent immediate, IS- Intermittent 24 hr survival.

Exposure	Myelin disruption %
Sham, 4hrs, CI	5.0 ± 0.6
60Hz, 1hr, CI	$15.6 \pm 2.2^*$
30Hz, 4hr, CI	$24.5 \pm 3.4^*$
120Hz, 4hr, CI	$28.0 \pm 1.7^*$
800Hz, 4hr, CI	$16.9 \pm 1.6^*$
60Hz, 4hr, CI	$28.6 \pm 1.8^*$
60Hz, 4hr, CS	$36.2 \pm 1.8^*$
60Hz, 4hr, II	$47.7 \pm 1.9^*$
60Hz, 4hr, IS	$45.3 \pm 5.7^*$

Discussion

1. Vibration exposure duration as short as 1 hr at 60 Hz can cause myelin disruption.
2. Damage is not limited to a single frequency.
3. Frequent rest periods do not reduce, but exacerbate, damage as evidenced by increased myelin disruption and transient hypersensitivity.

References

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2. Curry BD, Bain JL, Yan JG, Zhang LL, Yamaguchi M, Matloub HS, et al. Vibration injury damages arterial endothelial cells. *Muscle Nerve* 2002; 25:527-534.

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