ASSESSING IMPACT-TOOL VIBRATION DAMAGE OF TISSUES IN A RAT-TAIL MODEL

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1. INTRODUCTION

The risk of Hand Arm Vibration Syndrome (HAVS) has been linked to years of using vibrating powered tools with dominant frequencies in the 30-250 Hz range. The current International Standard ISO 5349 defines vibration risk exposure in the workplace based on frequency-weighted acceleration which diminishes risk for frequencies above 16 Hz. Impact tools, like riveting and chipping hammers, deliver shock waves with kHz frequency power superimposed on the 30 to 90 Hz duty cycles [1]. The high frequency content of the shock waves is nullified by frequency weighting, but are these shock waves harmful? McKenna etal. reported that the worker holding the metal bucking bar against the rivet is 4 times more likely to develop vibration white finger than the worker operating the rivet gun [2]. This suggests that tissue damage is occurring because the hands of the bucking bar worker are exposed to transmitted shock wave vibration. Previously, we developed a rat-tail vibration injury model to test the effects of sinusoidal vibration in the 30 to 800 Hz range [3]. The sinusoidal vibration was delivered by a B&K 4809 motor. In the present study, an Atlas Copco riveting hammer (RRH04P) accelerated a fan-shaped, steel tool bit that served as the vibration platform (Fig. 1). The rat tail is taped to the impact platform to model the worker's hand in contact with the bucking bar. We predicted that the degree of functional and structure damage following shock wave vibration would be much greater than that generated by the lower frequency content of sinusoidal acceleration.

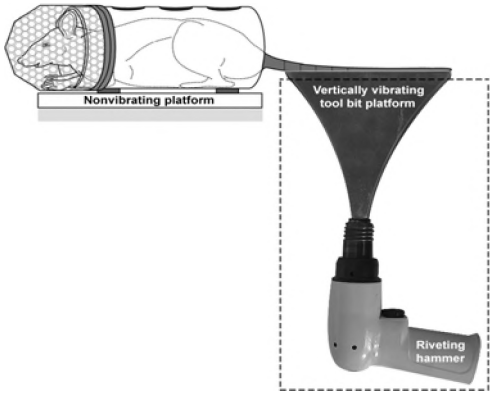

Figure 1. Rat-tail impact vibration model. The tail is taped to the fan-shaped, steel tool bit to simulate vibration from a bucking bar. The bit is accelerated vertically by the riveting hammer enclosed within a sound dampening box (dotted line).

2. METHODS

Male Sprague-Dawley rats (275-300 g) were divided into 4 groups (n=8/group): 1) impact vibration immediate (0-day recovery), 2) vibration 4-day recovery, 3) immediate sham control and 4) 4-day sham control. These procedures were approved by the Institutional Animal Care and Use Committee of the Medical College of Wisconsin.

Awake rats were restrained in tubes mounted to a non-vibrating platform, and the tail was taped to the riveting hammer tool bit (Figure 1). The riveting hammer was activated by 20 psi air, and the duty cycle was 33 Hz. Vibration exposure was 12 minutes continuous to simulate an intense period of using a bucking bar. The sham control rats were restrained in tubes, and the tails were taped to nonvibrating platforms. After vibration, the immediate 0 day groups were tail flick tested by measuring the tail withdrawal response time to noxious heat exposure. The rats were then deeply anesthetized and euthanized. The ventral nerve trunks and proximal tail skin were removed and processed for light and electron microscopy. The innervation of the skin was visualized by PGP9.5 immunoreactivity. Mast cells were stained with avidin-fluorophore conjugate.

The 4-day recovery, vibrated rats and the sham controls were returned to their cages. Tail flick testing was performed on days 2 and 4. On day 4, the recovery rats were euthanized, and the tail tissues were processed as described for the immediate groups. Tail flick responses were compared by a two-way repeated measures analysis of variance for treatment times day. Significance was accepted at $p < 0.05$.

3. RESULTS

Tail flick response times, measured before vibration treatment, were similar for the vibration and sham groups. Tail flick times for the vibration immediate rats were 34% shorter than the pre-vibration values ($p < 0.01$). The sham controls were unchanged from the pre-vibration values. By day 4 of recovery, the vibration groups exhibited a 32% prolongation of tail flick withdrawal time ($p < 0.001$).

Electron microscopy of the nerve trunks revealed the presence of axons with disrupted myelin in the immediate and 4-day vibration recovery groups (Figure 2). Evidence for loss of myelinated and nonmyelinated axons was not observed in the vibrated groups.

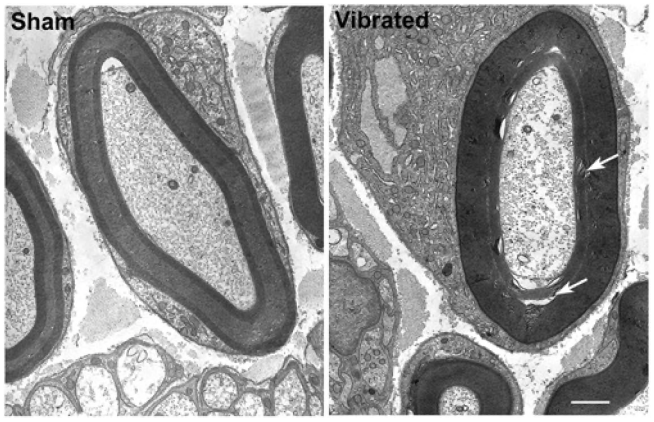

Figure 2. Vibration causes myelin delamination (arrows). White bar equals 1 micron for both panels.

Compared to the sham control, the terminal nerve fibers revealed by PGP9.5 immunoreactivity in the dermis of the skin were fragmented immediately and 4 days after vibration (Figure 3).

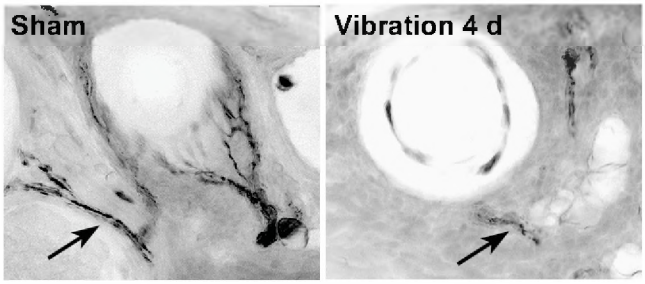

Figure 3. Intact PGP9.5 immunoreactive nerve fibers (left arrow) in the dermis of the sham. Vibration causes fragmentation and loss of nerve fibers (right arrow).

Mast cells are numerous in the dermis of the skin. Degranulation of mast cells was uncommon in the sham. Immediately after vibration (0 d), mast cell degranulation was pronounced (Figure 4). By 4 days, mast cell degranulation was similar to that in the sham control.

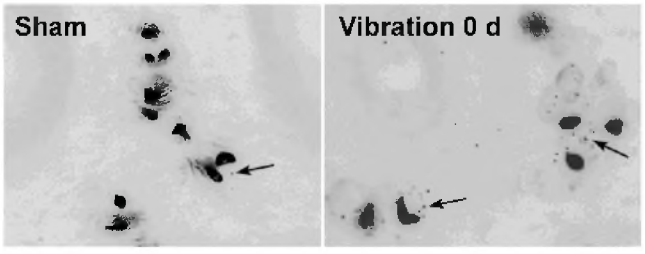

Figure 4. Mast cells in the dermis of the Sham exhibit occasional secretory granules (arrow). Immediately following vibration (0 d), many mast cells have released secretory granules (arrows).

4. DISCUSSION AND CONCLUSIONS

Exposure of the rat tail to a single period of impact shock vibration produces profound changes in the function and structure of the innervation. Immediately after vibration, the tail skin is hyper sensitive to noxious heat stimulation. By 4 days after vibration, the prolonged response to heat shows hypo sensitivity. The shift from hyper to hypo sensitivity does not correlate with the occurrence of axons with disrupted myelin in the nerve trunks. There is also no loss of axons in the nerve trunks to explain the altered responses to noxious heat. However, the fragmentation of PGP9.5 immunoreactive nerve fibers in the skin suggests that the immediate damage to nerve fibers renders them hyper sensitive to sensory stimuli. Nerve fiber hyper sensitivity is exacerbated by mast cell secretion of histamine and other inflammatory factors [4]. By 4 days, the damaged nerve fibers appear as discontinuous clumps, indicating necrosis and phagocytosis. The loss of terminal nerve fibers accounts for the hypo sensitivity to heat stimulation.

The intact parent axons in the nerve trunks can regenerate nerve endings. Regeneration was observed at 2 weeks after shock wave damage of skin with a lithotripsy machine [5]. Failed regeneration from repeated vibration injury may explain the persistent neuropathy in HAVS.

Further studies are necessary to determine the causes of nerve damage and mast cell degranulation. The major factors are the high frequency content and high acceleration levels of the impact shock vibration. The present findings indicate that frequency weighting under-estimates the risk of nerve damage in workers using impact hand tools.

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