

Keynote Presentation

SOME ASPECTS OF PATHOGENESIS OF VIBRATION-INDUCED WHITE FINGER

Hisataka Sakakibara
Nagoya University School of Health Sciences, Nagoya, Japan

Introduction

Although the pathophysiology of vibration-induced white finger (VWF) is still under discussion, evidence has been accumulated to understand the underlying mechanism.

VWF is pathophysiologically characterized by an enhanced vasospastic response to cold, which can result from an imbalance between vasoconstriction and vasodilation in the digital arteries in response to cold (i.e., vasoconstriction-dominant). The imbalance is supposed to be due to faults in vascular vessels and sympathetically mediated vascular tone.

Enhanced vasospastic response to cold

Structural factors for enhanced vasoconstriction (and vasodilation)

- Narrowing of arterial lumen with medial smooth muscle hypertrophy.

Possible functional factors for enhanced vasoconstriction

- Increased sympathetic nervous activity to cold (e.g., norepinephrine)
- Increased release of endothelin-1 (ET-1; an endothelial-dependent vasoconstrictor) from the endothelium
- Increased reactivity of alpha2-adrenoreceptors to cold

Possible functional factors for decreased vasodilation

- Decreased release of nitric oxide (NO; an endothelial-dependent vasodilator) from the endothelium
- Decreased release of calcitonin gene-related peptide (CGRP; a vasodilatory neuropeptide) from sensory afferents

The question is how their interrelations or imbalances among them are.

Vibration and arterial damage

The next question is, how does hand-arm vibration exposure induce such pathophysiological changes in VWF patients? Recent morphological evidence from animal experiments shows that vibration acceleration stress (including shear stress) and smooth muscle contraction contribute to arterial damage of smooth muscle and endothelial cells. The vibration-induced arterial damage is frequency-amplitude-dependent.

Repeated vibration exposure may damage smooth muscle cells to medial hypertrophy leading to lumen narrowing and injure endothelial cells to impaired vasodilation, resulting in vasospastic response to cold. The enhanced vasospastic response might in turn exaggerate vasoconstriction in response to cold.

References

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